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ELECTROMYOGRAPHIC INVESTIGATION OF INTRINSIC LARYNGEAL MUSCLES IN HUMANS

BY

K. FAABORG-ANDERSEN

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IN

From the Institute of Neurophysiology
University of Copenhagen, Denmark

ELECTROMYOGRAPHIC INVESTIGATION OF INTRINSIC LARYNGEAL MUSCLES IN HUMANS

An investigation of subjects with
normally movable vocal cords and patients with
vocal cord paresis

BY

K. FAABORG-ANDERSEN

COPENHAGEN 1957

Denne afhandling er
af det lægevidenskabelige fakultet
ved Københavns universitet
antaget til offentlig at forsvares for
den medicinske doktorgrad.

København, den 8. februar 1957.

KNUD BRØCHNER-MORTENSEN
h. a. dec.

S. L. MØLLERS BOGTRYKKERI. KØBENHAVN

To my best Friend – my Wife

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PREFACE

The investigations presented here were carried out at the Institute of Neurophysiology of the University of Copenhagen whose director Professor Fritz Buchthal M.D. with his great experience, critical sense, his scientific knowledge and his personal helpfulness has been of inestimable value for the completion of this work in all its phases.

The study was carried out in the years 1952–1956 during my appointment as I. physician at the ear, nose and throat department of the Finsen Institute and the ear, nose and throat department of Frederiksberg Hospital.

My former chief, Helmer Rasmussen M.D., who aroused my interest for the pathogenesis of vocal cord pareses has given me advice and guidance and offered me unusually fine working conditions.

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Mrs. A. Guld M.A. has given of her experience as speech therapist in instructing the subjects before the experiments.

Margaret Lennox M.D. has translated the manuscript to English.

The work has been supported by grants from the Danish State Research Foundation and the P. Carl Petersen Foundation.

I wish to express my deep gratitude to all who have assisted me,

those named and unnamed. Particularly I would like to thank Professor K. Goerttler M.D., J. van den Berg M.D. and J. Moll M.D. for kindly allowing me to reproduce illustrations from their books and papers.

Finally I would like to thank my children for the touching acquiescence with which they have relinquished me for the sake of this work.

Frederiksberg, February 1957.

K. FAABORG-ANDERSEN

Statement of the Problem and Review of the Literature

I. STATEMENT OF THE PROBLEM

The intrinsic laryngeal muscles have two functions: to move the vocal cords and to fix the vocal cords at different lengths, that is at different degrees of stretch.

As early as 1837 *Johannes Müller* showed that the pitch of the tone produced in the excised human larynx depended on the degree of stretch of the vocal cords. Recent stroboscopic and cinematographic investigations in human subjects have clarified the movements of the vocal cords associated with phonation but have not contributed information as to which intrinsic laryngeal muscles are active in producing these movements nor which are active in fixing and stretching the vocal cords.

Such information may be obtained by electromyographic investigations. It was the aim of the study presented here to determine the degree of activity of the intrinsic laryngeal muscles in normal subjects at rest, during respiration and especially during phonation. In this way indirect information can be obtained as to which muscles serve to move and stretch the vocal cords. In addition in an attempt to clarify its pathogenesis patients with unilateral or bilateral vocal cord paresis were investigated.

II. MOVEMENTS OF THE VOCAL CORDS IN THE NORMAL LARYNX

a) **Anatomical Introduction**

1) *The Function of the Intrinsic Laryngeal Muscles*

In contrast to the extrinsic laryngeal muscles, which move or fix

the whole larynx, the intrinsic laryngeal muscles serve to move the vocal cords. They are the cricothyroid, thyro-arytenoid, vocal, transverse and oblique arytenoid and lateral and posterior crico-arytenoid muscles (Table 1).

Table 1

Synonymous Terms for the Intrinsic Laryngeal Muscles

Designation of muscles in text	Designation of muscles in »Nomina Anatomica« 1955	Former designation of muscles
Cricothyroid muscle	M.cricothyreoideus	M.crico-thyreoideus externus
Vocal muscle	M.vocalis	M.thyreo-arytenoideus internus
Thyro-arytenoid muscle	M.thyreo-arytenoideus	M.thyreo-arytenoideus externus
Transverse arytenoid muscle	M.arytenoideus transversus	M.interarytenoideus transversus
Oblique arytenoid muscle	M.arytenoideus obliquus	M.interarytenoideus obliquus
Lateral crico-arytenoid muscle	M.crico-arytenoideus lateralis	M.crico-arytenoideus lateralis
Posterior crico-arytenoid muscle	M.crico-arytenoideus posterior	M.posticus

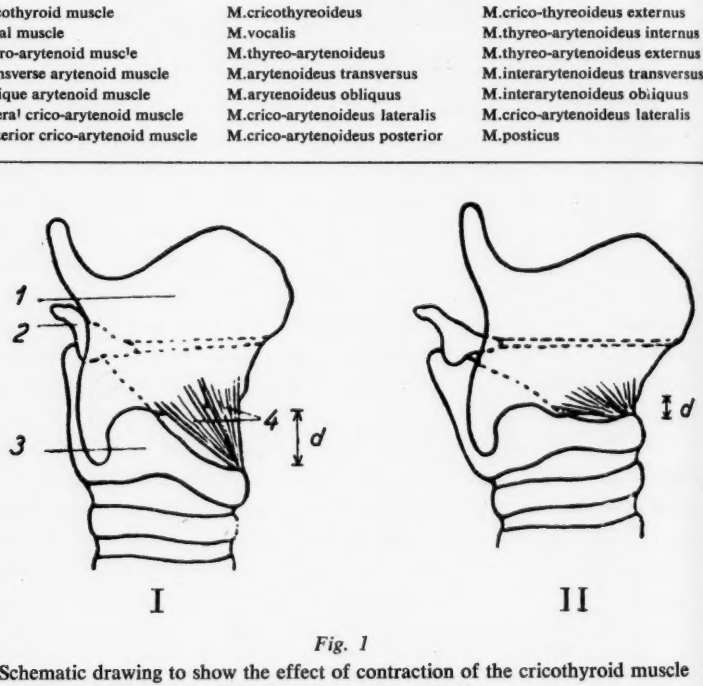

Fig. 1

Schematic drawing to show the effect of contraction of the cricothyroid muscle on the vocal cords (Modified after *Logan Turner* 1936).

1. Thyroid cartilage.
2. Arytenoid cartilage.
3. Cricoid cartilage.
4. Cricothyroid muscle.

d. Distance between the arch of the cricoid cartilage and the inferior border of the thyroid cartilage.

I. During relaxation of the cricothyroid muscle.

II. Contraction of the cricothyroid muscle.

In addition certain muscles within the larynx neither move the vocal cords nor move nor fix the larynx itself but act as accessories in closing the larynx: the thyro-epiglottic, ary-epiglottic and ventricular muscles.

The function of the *cricothyroid muscle* is to adduct and to stretch the vocal cords. Its contraction draws the bow of the cricoid cartilage closer to the anterior inferior edge of the thyroid cartilage. As a result the posterior superior edge of the cricoid cartilage, and the arytenoid cartilages attached to it, are rotated posteriorly (Figs. 1 and 4), thus adducting and stretching the vocal cords. (*Longet* 1869, *Schmidt* 1873, *Schech* 1873, *Rühlmann* 1874, *Hooper* 1883 and 1885, *Jelenffy* 1888, *Mullin* 1928, *Lemere* 1933, *Freedmann* 1956). There seems to be some individual variation as to whether the cricoid cartilage is moved towards the thyroid cartilage or vice versa, apparently dependent on which cartilage is fixed. In most instances the thyroid cartilage is fixed by the extrinsic laryngeal muscles and it is the bow of the cricoid cartilage which moves (*Hooper* 1883).

The distance ("d", Fig. 1) between the bow of the cricoid and the inferior edge of the thyroid cartilage was measured on X-ray photographs of normal subjects at rest and during phonation of tones of different pitch (*Möller* and *Fischer* 1904). The distance was greatest at rest and decreased progressively with increasing tone pitch by as much as 50 per cent indicating a progressive shortening of the cricothyroid muscle. Since the arytenoid cartilage is fixed to the cricoid cartilage, it follows that the vocal cords must be stretched during phonation of tones of increasing pitch. A progressive lengthening of the vocal cords by at most four mm (or about 25 per cent) during phonation of tones of increasing pitch was found by measurement of X-ray photographs of the larynx from the lateral aspect (*Sonninen* 1954).

When the *thyro-arytenoid muscle* (Fig. 4) contracts the arytenoid and thyroid cartilages are drawn closer to each other resulting in an adduction of the vocal cords, in their shortening and in an increase of tension (*Musehold* 1898, *Rühlmann* 1874, *Woods* 1893).

The function of the *vocal muscle* is generally considered to be the same as that of the thyro-arytenoid muscle. Recent investigations of the course of fibres in the vocal muscle have thrown doubt on this view. *Goerttler* (1950) demonstrated that the majority of fibres do

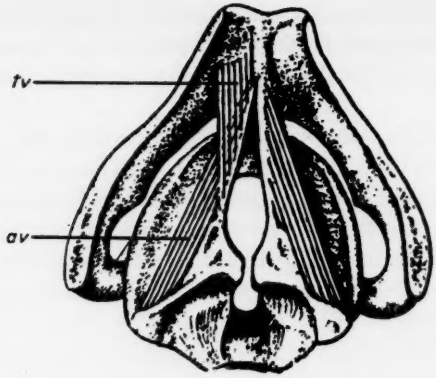

Fig. 2

Horizontal transection of the larynx. Both vocal cords and the glottis viewed from above. The fibre course in both aryvocal muscle (av) and in the left thyrovocal muscle (tv) is shown (*Goerttler* 1950).

not extend from the thyroid to the arytenoid cartilage as was assumed but that this muscle consists of a complicated system of fibres (Fig. 2). Two main bundles can be distinguished. One extends from the thyroid cartilage obliquely and posteriorly to insert into the vocal cord itself (the thyrovocal muscle, tv in Fig. 2). Another main bundle extends from the arytenoid cartilage obliquely and anteriorly and also inserts into the vocal cord (the aryvocal muscle, av in Fig. 2). These two main bundles course across each other. Such a fibre arrangement should allow this muscle to influence the position and tension of the vocal cords in many different ways, according to whether the muscle bundles contract singly or en masse. The most recent investigations of the vocal muscle (*Wustrow* 1952, *Mayet* 1955, *Berg* and *Moll* 1955) do not confirm *Goerttler*'s description in detail but indicate that though there are indeed crossed fibres inserted directly into the vocal cords, the main fibre bundle extends from the thyroid cartilage to the arytenoid cartilage (Fig. 3). However, the discussion as to the fibre course of the vocal muscle continues (*Behringer* 1954–55).

The transverse and oblique arytenoid muscles (Fig. 4) effect by their contraction an apposition of the posterior edges of the two arytenoid cartilages and thus assist in closing the cartilaginous portion of the glottis (*Luschka* 1871, *Vierordt* 1877).

Fig. 3

The course of the main portion of the fibres of the vocal muscle (*Berg* and *Moll* 1955).
St. Arytenoid cartilage.
S. Thyroid cartilage.
TV. Thyro-vocal portion.
TM. Thyro-muscular portion.

Contraction of the *lateral crico-arytenoid muscle* draws the vocal process of the arytenoid cartilage to the midline (Fig. 4) thus helping to close the glottis (*Hooper* 1892). There is, however, some evidence that certain bundles of this muscle may abduct the vocal process (*Rühlmann* 1874, *van den Berg* and *Moll* 1955).

The effect of contraction of *the posterior crico-arytenoid muscle* is to draw the vocal process of the arytenoid cartilage laterally (Fig. 4) and thereby to open the glottis (*Longet* 1869, *Schmidt* 1873, *Schech* 1873, *Hooper* 1892, *Klempener* 1899). It has also been claimed that the effect of this muscle in fixing the arytenoid cartilage provides the other muscles with a steady fulcrum and thus indirectly contributes to increasing the tension of the vocal cords (*Jelenffy* 1888, *Pressman* 1942, *Negus* 1947, *Jeschek* 1953). This contention has, however, not been confirmed in the present study (p. 54).

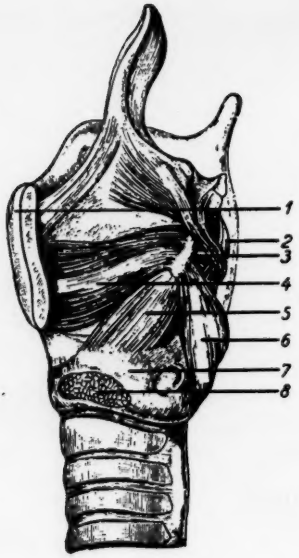

Fig. 4

The intrinsic laryngeal muscles seen from the lateral view after removal of one half of the thyroid cartilage (Modified after *Denker* and *Albrecht* 1932).

1. Thyroid cartilage.
2. Oblique arytenoid muscle.
3. Transverse arytenoid muscle.
4. Thyro-arytenoid muscle.
5. Lateral crico-arytenoid muscle.
6. Posterior crico-arytenoid muscle.
7. Cricoid cartilage.
8. Cricothyroid muscle.

In summary adduction of the vocal cords is brought about by contraction of the cricothyroid, thyro-arytenoid, vocal, transverse and oblique arytenoid and of the lateral crico-arytenoid muscles. These are therefore often called the adductor muscles, in contrast to the only intrinsic laryngeal muscle whose contraction produces abduction of the vocal cords, the posterior crico-arytenoid muscle.

2) *The Innervation of the Intrinsic Laryngeal Muscles*

The literature is extensive on the innervation of different intrinsic laryngeal muscles as determined both by stimulation and by acute

and chronic denervation studies. It has long been evident that the recurrent nerve through one of its terminal branches, the inferior laryngeal nerve, was of decisive importance for the innervation of the laryngeal muscles (*Navratil* 1871). Later investigations demonstrated the importance of the superior laryngeal nerve (*Exner* 1884) especially for increasing the tension of the vocal cords (*Schech* 1873, *Katzenstein* 1892, *Wagner* 1892, *Grabower* 1898).

Strangely enough the innervation of the intrinsic laryngeal muscles was investigated first in experimental animals and it was not until later that dissection of human cadavers demonstrated that only the cricothyroid muscle is innervated through the external branch of

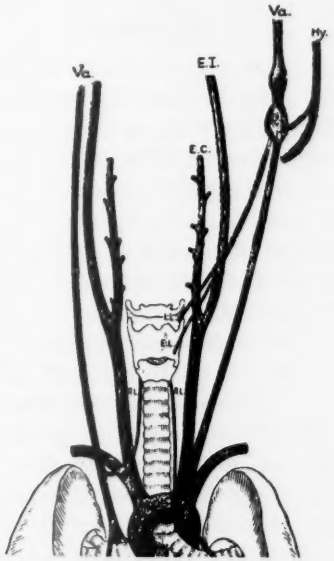

Fig. 5

Course of the superior and inferior laryngeal nerve (Adapted from *Cunningham* 1931).

Va. Vagus nerve.
Hy. Hypoglossal nerve.
I. L. Superior laryngeal nerve, internal branch.
E. L. Superior laryngeal nerve, external branch.
R. L. Inferior laryngeal nerve.
E. I. Internal carotic artery.
E. C. External carotic artery.

the superior laryngeal nerve. Most observations indicate that the arytenoid muscle is innervated both by the internal branch of the superior laryngeal nerve and by the inferior laryngeal nerve. All the rest of the intrinsic laryngeal muscles are innervated solely by the inferior laryngeal nerve (*Dilworth* 1921–22, *Berlin* and *Lahey* 1929, *Nordland* 1930, *Ziegelmann* 1933, *Weeks* and *Hinton* 1942, *Vogel* 1952). In many instances the inferior laryngeal nerve divides outside the larynx into two or more branches to the different intrinsic laryngeal muscles (*Reed* 1943, *King* and *Gregg* 1948, *Armstrong* and *Hinton* 1951, *Rustad* and *Morrison* 1952, *Morrison* 1952). Fig. 5 shows the course of the superior and inferior laryngeal nerves.

b) **Respiration**

During quiet respiration the vocal cords are in a position intermediate between full adduction and full abduction (Fig. 7). In most subjects the size of the glottis is the same during quiet inspiration as during quiet expiration (*Barwell* 1905) but in about 20 per cent of individuals the vocal cords move somewhat about the intermediate position, the glottis being larger during inspiration than during expiration (*Semon* 1890). During forced respiration there is always an enlargement of the glottis during inspiration and a narrowing during expiration (*Weiss* 1931).

These movements of the laryngeal muscles with respiration are thought to depend on muscular activity, but of which muscles is not certain. *Schech* (1873), *Rühlmann* (1874), *Bosworth* (1880) and *Semon* (1890) considered contraction of the posterior crico-arytenoid muscle to be mainly responsible. *Grossmann* (1898) considered an adductor relaxation during inspiration to be the cause of these movements. *Jelenffy* (1888) believed that the movements of the laryngeal muscles with respiration depended on an alternating contraction of the thyro-arytenoid and of the vocal muscles.

An electromyographic investigation of the intrinsic laryngeal muscles during respiration in cats has demonstrated an increase in activity of the posterior crico-arytenoid muscle during inspiration and of the vocal and lateral crico-arytenoid muscles during expiration (*Green* and *Neil* 1955). In rats activity during inspiration was demonstrated in, among others, the cricothyroid muscle but no activity during expiration (*Andrew* 1955). In humans electromyo-

graphic investigations have given conflicting results. According to *Weddell, Feinstein* and *Pattle* (1944) and *Feinstein* (1946) there is an increase in activity during inspiration and a lesser increase in activity during expiration in many of the internal laryngeal muscles, both in normals and in patients with laryngeal paresis. Other investigators have reported a greater increase in activity during expiration and a lesser increase during inspiration in the thyro-arytenoid muscle (*Husson* 1955) or in the cricothyroid and vocal muscles (*Fink, Basek* and *Epanchin* 1956).

c) Movement of the Vocal Cords During Phonation

1. *Spoken and Singing Voice*

At about the turn of the century the mechanism of voice production in the larynx was assumed to be as follows. When the vocal cords are adducted and tightened strong expiration blows them apart. On account of their elasticity they recoil and close off the stream of air, only to be forced apart again by expiratory pressure. In this way there occurs a rhythmical escape of puffs of air, or in other words there is produced a tone. It is not the vibration of the vocal cords themselves which produces the tone but the fact that they interrupt the stream of air (*Ewald* 1898, *Musehold* 1898).

Even if the major vibrations of the vocal cords occur in the horizontal plane, there are also smaller vibrations in the vertical plane (*Rethi* 1896, *Musehold* 1898) in that the vocal cords open with an upward movement and close with a downward movement.

The range of the singing voice is divided into different registers (Fig. 6). By register is meant that portion of the tone scale which a given singer can sing without changing the quality, that is the number of overtones, of the single tones. Opinons differ among laryngologists and phoneticists as to the number of registers in men and women. Three main registers are differentiated: the chest register includes the lower tones with many overtones; the middle register includes somewhat higher tones with fewer overtones; and the head register occupies the upper end of the tone scale where the tones have the smallest number of overtones (*Donders* 1858, *Katzenstein* 1911, *Sokolowsky* 1913). Above the head register there is an "upper thin" register, and below the chest register in men there may be a "bass" register. Most investigators agree that all three main registers may

be found in women. Some believe that men also have three registers, while others claim that men usually only have chest and head registers.

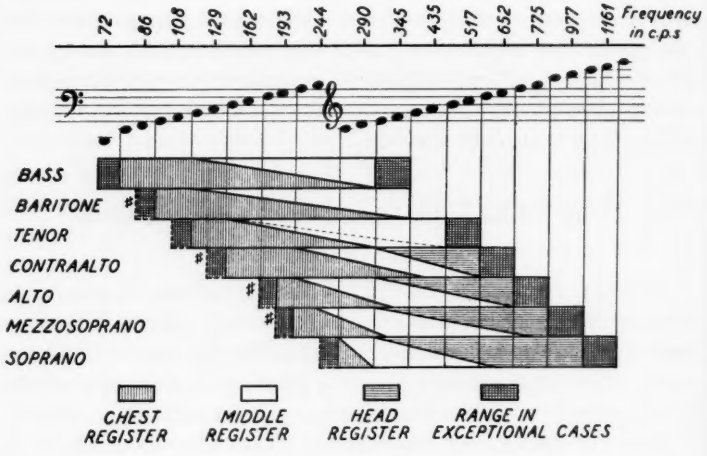

Fig. 6
Frequency and register range for different voices (Modified after *Weiss* 1931).

If one listens to an untrained singer perform a scale, one can clearly hear the shift from one register to the other. The change in register does not occur at the same place in the scale in all singers; partly it is different according to whether the voice is bass, tenor, alto or soprano; and partly there are individual variations within these groups. Overtones arise in glottis itself as a result of the vibrations which produce the explosive puffs of air here (*Johannes Müller* 1837, *W. Trendelenburg* 1935, *Pressman* and *Kelemen* 1955) and are therefore not a resonance phenomenon.

During the early investigations of the movements of the vocal cords during phonation on the excised human larynx (*Johannes Müller* 1837), and especially during the early stroboscopic investigations (*Musehold* 1898), it was noticed that the glottis had a different appearance when tones in the different registers were being produced. With tone production in the chest register both vocal cords lay in close apposition to each other and vibrated over their whole length. With increasing pitch from middle to head register the glottis became

wider and more spindleshaped, so that the vocal cords only occasionally touched each other, and only the edges of the vocal cords oscillated (*W. Trendelenburg* and *Wullstein* 1935, *Luchsinger* 1949).

Since the pioneer investigations of *Johannes Müller* more than one hundred years ago, the extirpated larynx of human cadavers has been used to investigate the effect of changes in the vocal cords on the quality of tones produced. It has been found that the pitch of the tone is dependent in the first instance on the tension of the vocal cords, in that the pitch increases with increasing tension; while the intensity of the tone is dependent mostly on the pressure of air in the trachea, in that the tone intensity increases with increasing air pressure (*Johannes Müller* 1837, *Wyllie* 1866, *Jelenffy* 1888, *Wethlo* 1949). These observations formed the basis of the "myoelastic theory" as to the formation of tones in the larynx.

The newer "aerodynamic theory" is based on further observations of the behavior of the vocal cords during speech. During phonation the vocal cords have two lips, an upper and a lower, and the lower lip is both abducted and adducted before the upper (*Bell Telephone Laboratories'* High Speed Motion Film 1940, *Smith* 1954). These movements are seen only during phonation of tones in the range of speech. Furthermore it became apparent that the recoil of the vocal cords can more probably be attributed to the suction which occurs as a result of the passage of a stream of air through the narrow portion of a tube (*Tonndorf* 1925, *Smith* 1954). If the walls of the tube are elastic, they will be drawn in by this suction.

A new theory has been advanced by *Husson* (1951) to explain tone production in the larynx. According to this "neuro-muscular theory" the frequency of vibration of the vocal cords at a given pitch is the direct result of a corresponding number of impulses received from the nerves innervating these muscles. According to *Husson* the upper vibrating frequency of the vocal cords is limited by the refractory period of the nerves which he assumes as 2 msec. Accordingly the vocal cords cannot vibrate at a rate greater than 500 cycles per second. In order to explain the production of high pitched tones *Husson* assumes that for tone frequencies between 500 and 1000 cycles per second the single muscle fibres receive impulses only at every other movement of the vocal cords, in other words, one half of the muscle fibres are innervated for one movement of the vocal cords, and the other half for the next, and so on. For

tones higher than 1000 cycles per second the single muscle fibres are innervated similarly only at every third movement of the vocal cords. These conditions, according to *Husson* (1952), explain the changes which occur with a shift in register.

Husson cites *Erlanger* and *Gasser* as having determined the absolutely refractory period of nerves be 2 msec. Actually *Erlanger* and *Gasser* (1937) demonstrated a refractory period for the A-fibres, including the motor nerve fibres, of 0.4–1 msec. On the other hand the refractory period of nerve can only be the limiting factor if it is longer than the refractory period of muscle fibres. The absolute refractory period of muscle is 3–4 msec (*Buchthal* 1957 a). It would therefore necessarily be this time interval and not the refractory period of nerve which would determine the maximal tone frequency at which the muscle fibres are innervated with every movement of the vocal cords.

Lullies (1953) has pointed out that in order to evaluate the neuromuscular theory, investigations must be undertaken to determine whether action potentials can be found in the recurrent nerve of the same frequency as that of a simultaneously produced tone. *Moulonguet* (1955) has reported in a single subject during laryngectomy that there was an approximate agreement between the tone frequency and the frequency of the action potentials recorded from the recurrent nerve. However, these action potentials were measured in non-synchronized, not in synchronized, bursts and no conclusion can be drawn as to frequency in single nerve fibres. Similarly in experiments with dogs, *Lindemann* (1930) has reported action potentials in the recurrent nerve at a frequency of 380–1800 per second, corresponding to the tone produced. Since information is not given as to whether the action potentials were those of a single nerve fibre it must be assumed that also in this instance the frequency was measured in a non-synchronized bundle of fibres and that no conclusions can be drawn as to the frequency in single nerve fibres.

It should further be investigated whether the intrinsic laryngeal muscles can contract as rapidly as the assumed action potentials led from the recurrent nerve. There is somewhat contradictory evidence on this point. In dog experiments the exposed recurrent nerve was stimulated with a train of brief pulses (*Laget* 1953) and it was found that the vocal cords followed the frequency of the stimulating pulse to 450–500 pulses per second, when the vocal cord movements

ceased, to return again at higher stimulating frequencies. Similar investigations have demonstrated that the vocal cords' vibration frequency follows the stimulating frequency only at very low frequencies (7 pulses per second). At higher frequencies (12 pulses per second) the movements of the vocal cords become irregular and still higher frequencies result in a tetanic contraction of the vocal cord musculature (25 pulses per second, *Vallancien* 1953; 30–40 pulses per second, *Vallancien* 1955). Neither in dogs (*Amersbach* 1922) nor in humans (*Katsuki* 1950, present investigation) was it possible to demonstrate in the intrinsic laryngeal muscles action potentials of such a frequency as to even approximate that of the tone produced. *Husson* (1955) reports, however, that he has measured action potentials with a frequency up to 500 per second in the thyro-arytenoid and the vocal muscles in patients during laryngeal operations. Again these frequency measurements were undertaken in groups of muscle fibres whose action potentials were not synchronized and they do not therefore permit conclusions as to the frequency of discharge of single motor units.

In small insects (*Hymenoptera* and *Diptera*) whose wings oscillate at frequencies over 1000 cycles per second, action potentials have been led off from the indirect flight muscles at frequencies of 100–200 cycles per second (*Pringle* 1949). Such a myogenically determined rhythm has, however, never been reported in the skeletal musculature of vertebrates.

2) *Whispered Voice*

During the production of whispered tones the glottis can have different appearances; the Y-form is said to be especially characteristic. This form appears when the membranous portion of the glottis is closed while the cartilaginous portion remains open. It has been assumed that the cause of this is a powerful contraction of the lateral crico-arytenoid muscle (*Gutzmann* 1909) while the arytenoid muscle does not contract (*Pressman* 1942). This last assumption has, however, not been confirmed in the study presented here.

d) **Closure of the Laryngeal Aperture during Cough and Swallow**

With cough and swallow the laryngeal aperture is closed. It has been demonstrated in animal experiments, in which a T-tube was

placed in the trachea and the vocal cords were adducted by external pressure on the neck, that the ventricular folds close the larynx during cough while the inspiratory closure with swallow is effected by the vocal folds (*Wyllie* 1866, *Brunton* 1883) in that both the ventricular folds and the vocal folds function as valves which can be moved by air pressure alone without appreciable muscular exertion so as to close off the larynx (*Pressman* 1944).

Electromyographic investigations of the mechanism of swallow have been carried out in monkeys, cats and dogs (*Dotty* and *Bosma* 1956). The interactions of twenty-two different muscles in the mouth, pharynx and larynx were studied, among them the thyro-arytenoid, the arytenoid and the cricothyroid muscles. It was demonstrated that these muscles contract in the last phase of swallow at approximately the same time as the midportion of the constrictor muscle of the pharynx. The thyro-arytenoid and arytenoid muscles are the first to contract and are followed by the pharyngeal constrictor muscle and the cricothyroid muscle in that order. The exact time of contraction varied somewhat from animal to animal.

III. PARETIC VOCAL CORDS

Paresis of one vocal cord can only be due to paresis of the recurrent nerve in the absence of such mechanical causes as tumor of the larynx, cicatrix in the larynx or ankylosis of the crico-arytenoid articulation. The first laryngoscopically verified instances of recurrent nerve paresis were reported by *Türch* in 1860 and *Gerhardt* in 1863 who described the paretic vocal cord to remain motionless in the midline. It was assumed that the cause was a paresis of the intrinsic laryngeal muscles but it was not entirely clear which muscles were paralyzed. Since the patients' spoken voice was good it was suggested that the paresis involved the posterior crico-arytenoid muscle only (*Gerhardt* 1863, *Riegel* 1872) but the claim was also made that all the intrinsic laryngeal muscles were paralyzed (*Türch* 1860, *Penzoldt* 1874, *Bosworth* 1880).

At that time it was known that the recurrent nerve innervates the laryngeal abductor, the posterior crico-arytenoid muscle, as well as many of the laryngeal adductors. At about the same time a series of clinical observations demonstrated that the initial position of the vocal cord in recurrent nerve paresis caused by pressure on the nerve

was usually near the midline; only later in the course of the illness did it move more laterally to the intermediate or cadaver position. It was postulated therefore that the abductor fibres in the recurrent nerve are more apt to be affected than the adductor fibres so that a crico-arytenoid paresis occurs first and later a total recurrent paresis (*Rosenbach* 1880, *Semon* 1881, 1890, 1897, 1904; *Semon* and *Horsley* 1886, 1889, 1892; *Delevan* 1884, *Barwell* 1905, *Gleitsmann* 1908, *Casselberry* 1908).

This theory, the "*Semon-Rosenbach*'s law" was supported by numerous experimental observations on the effects of muscle transection and nerve stimulation in different animals, mostly dogs (*Hooper* 1885, 1887; *Katzenstein* 1892, 1903; *Grabower* 1898, 1904; *Kuttner* 1903, *Russel* 1892, *Fraenkel* and *Gad* 1889). Other experimental investigations of the same kind supplied evidence against it (*Krause* 1884, *Donaldson* 1886, *Wagner* 1890, 1892; *Grossmann* 1897). As the clinical observations accumulated there were more and more who felt convinced of the reality of "*Semon-Rosenbach*'s law" (*Saundby* and *Hewetson* 1904, *Critchly, Mac Donald* and *Kubik* 1925, *New* and *Childny* 1932, *Herzog* 1933, *Smith, Lambert* and *Wallace* 1933, *Burger* 1934, *Scheicher* 1937, *Clerf* 1953).

Semon and his school did not attribute to the cricothyroid muscle any importance for the position of the paretic vocal cord while other investigators asserted that the cricothyroid muscle was of decisive importance for this position (*Wagner* 1890 and 1892, *Grossmann* 1897). This is at present the most generally accepted explanation of the different positions of the vocal cord in vocal cord paresis and is supported by the main body of recent work on this subject (*Burger* 1934, *Hofer* and *Jeschek* 1940, *Murtagh* 1945, *Negus* 1947, *Murtagh* and *Campbell* 1952, *Pressman* and *Kelemen* 1955). If only the recurrent nerve is interrupted the vocal cord maintains a fixed paramedian position a few millimeters from the midline since the cricothyroid muscle is unaffected. It may perhaps be of importance for the position of the vocal cord that the arythenoid muscle according to the opinion of most authors is innervated both by the recurrent and by the superior laryngeal nerves (cf. p. 18). If both the recurrent and the superior laryngeal nerves are interrupted, all the intrinsic laryngeal muscles are paralyzed and the vocal cord assumes an immovable position in the intermediate position between full adduction and full abduction, more laterally than the paramedian

position. Laryngoscopy often reveals the immovable vocal cord in the intermediate position to be more bow-formed and shorter than in the paramedian position (Fig. 7).

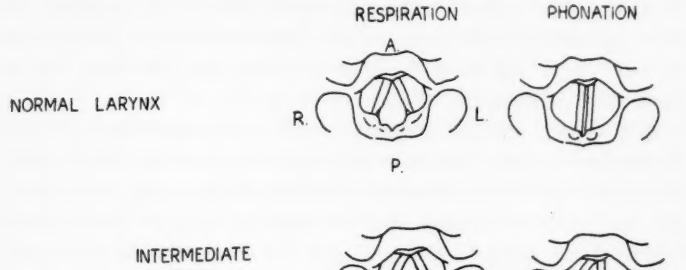

Fig. 7

Schematic drawing of the glottis as seen by indirect laryngoscopy at rest and during phonation in subjects with normally movable vocal cords (above) and in patients with unilateral vocal cord paresis in the intermediate position (in the middle) and the paramedian position (below).
R = right L = left A = anterior P = posterior

In the vocal cord pareses discussed above the paretic vocal cord is motionless; other vocal cord pareses (in different bilateral affections of the larynx) are characterized by a faulty adduction during phonation of the otherwise freely movable vocal cords (adductor, thyro-arytenoid and transverse paresis). These types of paresis are often seen in cachectic individuals and it is not known whether they are due to excessive fatigability of the muscles or whether they are in fact pareses.

Finally it should be mentioned that transection of the superior laryngeal nerve causes a relaxed vocal cord with scallopped edges without any alteration in the movability of the vocal cords (*Mygind* 1906, *Luchsinger* 1942).

IV. ELECTROMYOGRAPHY

Before the electromyographic findings in the laryngeal muscles, the findings will be briefly described in normal muscles and in muscles with neurogenic paresis. The following discussion is mainly derived from *Buchthal* (1957 b). In a relaxed muscle there is no demonstrable electrical activity under normal conditions. As the muscle contracts with increasing intensity action potentials from fibres of a given motor unit occur with increasing frequency. With weak contraction there are 3–4 action potentials per second, while with maximal contraction there may be 20–50 action potentials per second (*Adrian* and *Bronk* 1929). In the external ocular muscles, which are about the same size as the intrinsic laryngeal muscles, there may be up to 150–200 action potentials per second with maximal contraction (*Björk* and *Kugelberg* 1953, *Breinin* and *Moldaver* 1955).

With weak steady contraction the potential of a single motor unit can be recorded over long time periods. The frequency for such a motor unit is regular and an alternating appearance and disappearance of a motor unit is not seen (*Buchthal* and *Clemmesen* 1941). With maximal contraction, on the other hand, in the most excitable units, series of potentials alternate with periods of electrical silence. These silent periods can last as long as two seconds (*Norris* and *Gasteiger* 1955).

The intensity of muscle contraction is graded not only by the discharge frequency of the single motor unit but also by the number of innervated motor units. The strength of contraction can be inferred from the action potential recording: the mean amplitude of the integrated action potentials is directly proportional with the strength of contraction (*Wegener* 1941, *Lippold* 1952, *Inman, Ralston, Saunders, Feinstein* and *Wright* 1952).

In a normal muscle it is only possible to follow the activity of single motor units during weak muscular contraction. With stronger activity the recording shows many asynchronous action potentials and it is no longer possible to identify the single potentials of a given motor unit because of interference from the many activated fibres from different motor units in the neighbourhood of the electrode. Between the pattern of single motor unit discharges and the interference pattern there are all gradations; motor unit potentials from fibres close to the electrode can be identified because their amplitude

is higher than of potentials which arise from more distant fibres (*Buchthal* and *Clemmesen* 1941).

During recording from different points in the same muscle a wide variation is found in the amplitude, duration and form of the motor unit potentials (*Buchthal* and *Clemmesen* 1941, *Petersén* and *Kugelberg* 1949, *Buchthal, Guld* and *Rosenfalck* 1954). The variation in amplitude is due mainly to differences in the distance between electrode and active fibres (*Buchthal* and *Pinelli* 1951, *Buchthal, Guld* and *Rosenfalck* 1956), and is so large that deviations from normal must be considerable to be statistically significant. The variation in duration of action potentials is due partly to differences in the extent of the innervation zone for the different motor units and partly to differences in the position of the recording electrode relative to the innervation zone (*Buchthal, Guld* and *Rosenfalck* 1955). The mean duration is somewhat different in different muscles (*Buchthal* and *Rosenfalck* 1955). For small muscles such as the intrinsic laryngeal muscles the duration varies between 3 and 7 msec and at least 20 different potentials must be measured in order to determine the mean duration for any given muscle. The duration increases with age, being 75 per cent greater at 80 than at 1 year of age; and with falling temperature (*Bentsen* 1945, *Buchthal, Pinelli* and *Rosenfalck* 1954).

Action Potentials in Neurogenic Pareses

After total or partial denervation no muscle action potentials can be recorded from the fibres involved for 15–20 days. Then denervation or fibrillation potentials appear, spontaneous potentials of short duration (0.5–3 msec) and low amplitude (50–100 μV) and may remain for many years (*Denny-Brown* and *Pennybacker* 1938). During maximal voluntary effort after partial denervation, instead of the normal interference pattern, there appear only single motor unit potentials (*Buchthal* and *Clemmesen* 1941); mixed activity pattern (single units distinguishable against a background of interfering activity) is associated with mild neurogenic paresis.

There is an increase in action potential duration in one half to one third of patients with neurogenic paresis (*Buchthal* and *Clemmesen* 1941, *Buchthal* and *Pinelli* 1953, *Hertz, Madsen* and *Buchthal* 1954).

Material and Methods

I. SUBJECTS

The 32 subjects, 25 men and 7 women, who constitute the normal series were all healthy volunteers with normal larynx and freely movable vocal cords. In addition the normal or non-paretic side of the larynx was investigated in 23 patients, 10 men and 13 women, with unilateral vocal cord paresis. The age distribution of the subjects and of the patients is indicated in Fig. 8.

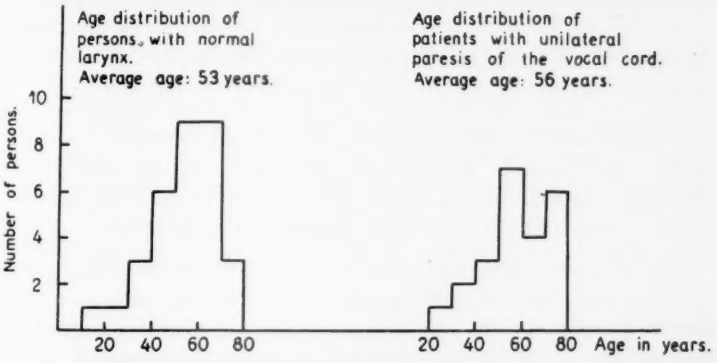

Fig. 8

Age distribution of normal subjects and patients with unilateral vocal cord paresis.

Only a few of the subjects were trained singers who without preceding exercises could vary pitch and intensity independently. Therefore a phoneticist instructed such exercises before each investigation. First the subjects were practiced in muscular relaxation during quiet respiration. Then intonation of the vowel "e" was practiced with medium and constant intensity and at a pitch approximately in the

middle of the voice range of that subject. The subject was then practiced in intonating the same vowel with increasing intensity at constant pitch; then two vowels of different pitch and the same intensity, as for example "ahe" where "e" was of higher pitch than "a". Further the subject was practiced in whispered voice and in "silent speech" – for the latter the patient was asked to think of phonating a given vowel without audible voice production. The subjects were further instructed in the use of different forms of tonal initiation or attack, as for example "coup de glotte" or stopped attack ("op"), even attack or static initiation ("bob") and breath attack ("hop"). They were instructed in intoning "op–bob–hop" sometimes in rapid, and sometimes in slow succession. In addition the subjects were asked to pronounce the same word with and without glottal stop or catch ("stød"), for example "mord" with "stød" and "mor" without "stød". Finally the subjects were practiced in glottal click which is produced when the glottis is closed and the air pressure in the trachea is increased to force the vocal cords apart with a single explosion without vocalization.

In addition the subject was instructed in ordinary respiration and forced respiration with activation of the auxiliary respiratory muscles.

II. EXPERIMENTAL ARRANGEMENT AND APPARATUS FOR RECORDING ACTION POTENTIALS FROM THE INTRINSIC LARYNGEAL MUSCLES WITH SIMULTANEOUS RECORDING OF THE SOUND PRODUCED

For local anesthesia the pharynx, hypopharynx and laryngeal mucosa of the subjects were sprayed with a solution containing 0.5 per cent tetracaine and 0.001 per cent adrenalin hydrochloride.

The subject was earthed through a plate electrode placed on a damp cloth on the back of the neck. In most experiments a concentric needle electrode (Fig. 10) was placed in the cricothyroid muscle by insertion through the skin on a level with the cricoid cartilage 1 cm lateral to the midline and held in place with adhesive tape (Fig. 9). By indirect laryngoscopy another concentric needle electrode was introduced through the open mouth and placed either in the vocal, the thyro-arytenoid, the arytenoid or in the posterior crico-arytenoid muscle. There were sometimes difficulties in placing the electrode in the posterior crico-arytenoid muscle, while place-

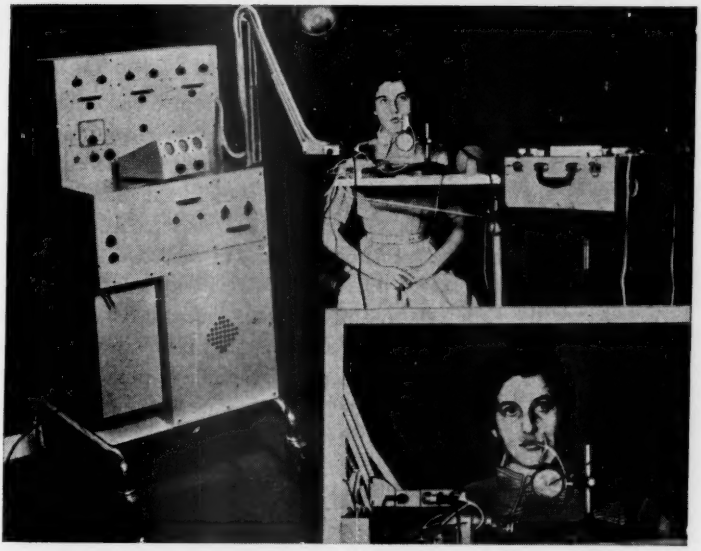

Fig. 9

Experimental apparatus and arrangement

To the left is seen the three channel electromyograph. To the right the subject and the tape recorder with microphone (F). On the table in front of the subject is seen the high-pass filter (A) inserted between the electromyograph and the electrodes. One needle electrode (B) is placed in the left cricothyroid muscle by insertion through the skin of the neck. Another needle electrode (C) is inserted through the mouth and placed in the vocal muscle. The third channel of the electromyograph is connected with a microphone (D) placed in front of the subject at a constant distance from the subject's mouth. A ground plate electrode (E) is attached to the back of the subject's neck.

ment in the other muscles was usually easy. Placement in the lateral crico-arytenoid muscle was still more difficult. It was probably possible on several occasions to record from this muscle. Since it was, however, impossible to determine with certainty that the electrode actually was in the lateral crico-arytenoid muscle, this muscle's activity was not systematically investigated. As for the arytenoid muscle it was not possible to differentiate needle placements in the transverse from those in the oblique arytenoid muscle. The depth of recording was 2–3 mm in all the muscles investigated.

Mucus in the larynx sometimes hampered electrode placement and could so compromise the quality of the tone produced as to

render difficult the determination of pitch. Therefore in some of the experiments atropine sulfate 0.5 mg was given by mouth one half hour before the investigation.

In the majority of investigations the electrode in the cricothyroid muscle was connected to one channel of the electromyograph and the electrode in the intrinsic laryngeal muscle to the second channel; exceptionally there were two needle electrodes in the same muscle at the same time. The output of a microphone was recorded on the third channel, so that the sound produced was recorded simultaneously with the muscle action potentials. In this way it was possible to investigate the time relationships between tone production and electrical activity of the different internal laryngeal muscles. In order to obtain a time correlation between the respiratory phases and the change in electrical activity an attempt was made to signal the onset of inspiration as determined by inspection and by palpation of the subject's thorax. The signal was sometimes a sound delivered to the microphone, sometimes a light mark on the photographic paper. In some cases the respiratory phases were recorded in addition with the help of a thermocouple in the subject's nose (*Arentsen* and *Kaiser* 1953).

In all instances the subject's voice was recorded on a tape recorder with a tape speed of 19 cm per second in order to record changes in register, pitch and intensity (Fig. 9).

Before and after each investigation the electrode placement in the laryngeal muscles was ascertained by indirect laryngoscopy; in addition in a number of subjects by inspecting the movements of the vocal cords during phonation during electromyographic recording. During such investigations the movement of the vocal cords in adduction and in abduction was signaled by saying "now" when the vocal cords began to move; this "now" was registered on the microphonic record together with the subject's phonation.

The muscle action potentials were recorded on photographic paper by an electromyograph (DISA) with three channels, each with its cathode-ray oscilloscope. Simultaneity was ascertained by introducing a common signal on the three channels. Parallel with these three cathode-ray oscilloscopes for recording were three other cathode-ray oscilloscopes for direct observation of the trace during or between recordings. In addition any one of the three channels could be connected with a loudspeaker. Each of the amplification

channels contained a four-stage differential amplifier with an input impedance of 100 megohms in parallel with 60 $\mu\mu$F. The noise level was 2 μV r.m.s. The frequency characteristic had fallen by 3 decibels at 3 and 10.000 cycles per second respectively (Fig. 9).

To eliminate the slow deflections of the base line arising from movements of the electrode due to phonation a high-pass filter was inserted between the electromyograph and the needle electrodes. The frequency characteristic of the filter had fallen by 3 decibels at 24 and 36 cycles per second respectively.

The recording on the electromyograph was either continuous with a paper speed of 5 cm per second or interrupted with single sweeps on the oscilloscopes with a speed of 1 mm per msec. The sweep frequency was five per second.

The interrupted recording was used partly to determine the tone frequency and partly to record the duration of the single muscle action potentials. To determine the tone frequency the mean duration of ten oscillations was measured. Calibration with a sine wave generator indicated that the frequency was measured with an accuracy of one per cent. In addition the frequency was determined of each sound recorded on the tape recorder. To this purpose the sound trace was played back into one beam of a cathode-ray oscilloscope and photographically recorded on continuously moving film (speed

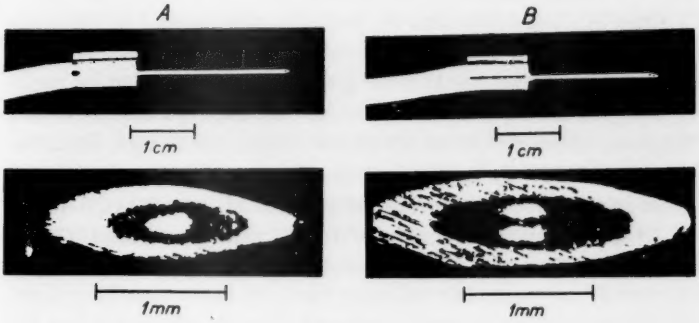

Fig. 10

Needle electrodes used for recording action potentials from the intrinsic laryngeal muscles.
A. Concentric electrode.
B. Bipolar electrode.
Above: needle electrodes with attachment for the laryngeal forceps.
Below: microphotograph of the electrode tips.

25 cm per second). A fifty cycle square wave signal was recorded on the other beam for calibration.

The concentric needle electrode used to record from the cricothyroid muscle was a steel cannula with an outer diameter of 0.55 mm containing an insulated 0.1 mm platinum wire. The potentials were measured between the tip of the platinum wire and the surrounding steel shaft. The tip angle of the cannula was 15–20°, so that the recording area of the platinum wire was 0.03 mm². The electrode impedance was between 100 and 200 k Ω at a measuring frequency of 150 cycles per second. To record from the intrinsic laryngeal muscles a modified form of the same electrode (Fig. 10) was used, or a bipolar electrode (Fig. 10) with two 0.1 mm insulated platinum wires in a steel cannula with an outer diameter of 0.65 mm. The angle of the electrode tip was 15–20°, and the platinum wires' terminal ends lay along the short axis of the ellipse. The recording area for each platinum wire was 0.03 mm². The distance between the midpoints of the two leading-off areas was 0.2 mm. The electrode impedance was between 100 and 200 k Ω at a measuring frequency of 150 cycles per second.

The electrodes were sterilized in water at 100° C for 20 minutes. An electric cooker was used which had a series of holes covered with rubber caps through which the needle electrodes could be inserted into the boiling water (*Buchthal* 1949).

Since in all investigations an electrode was inserted in the cricothyroid muscle through the external surface of the neck, a throat microphone was not used; rather phonation was recorded by means of a crystal microphone (*Ronette,* Amsterdam, type B 110), with a frequency characteristic of 30–10.000 cycles per second, linear between 40–1.000 cycles per second. The microphone amplitude is given in mV. 0.4 mV correspond to a sound intensity of 1 m bar or 74 decibels at a frequency of 1.000 cycles per second. (Sound Technical Laboratory, Royal Technical University of Denmark).

III. ELECTROMYOGRAPHIC CRITERIA

The amplitude of the action potential pattern was measured by tracing the envelope of the action potentials as recorded continuously at low speed and measuring the greatest height for each of the three to five successive phonations. The mean value in millivolts is termed

maximal amplitude in this report. The total duration of single action potentials on fast single sweep recording was measured as the time between the onset of the potential deflection and the point at which it returned to the base line (Fig. 15). When the degree of electrical activity was such that single motor unit potentials could be distinguished, their frequency of discharge was measured by measuring the distance between the single potentials in a series of action potentials from the same motor unit. The time relationship was determined on continuous recording between the onset of electrical activity and of phonation, respiration, swallowing and so forth.

The maximal amplitude of the action potential pattern depends in part on the distance between the active muscle fibres and the recording electrode, and in part on the degree to which the activity of many fibres is superimposed, reflecting the number of motor units activated.

In addition to recording the action potentials directly, the mean action potential amplitude was recorded by an electronic device *), whose output was proportional to the mean amplitude of the action potentials. In principle this was done by doubly rectifying the action potential and by then adjusting the rectified signal in a suitable way (Fig. 11). The action potentials were first led through a paraphase amplifier stage. The signals from the two anodes of the paraphase amplifier were identical in form and amplitude but reversed 180° in relation to each other. Each of the two signals was led through a cathode follower into its diode-detector which cut off the negative phase of the signal. By suitable choice of the time constant in the detector, dependent on the frequency of the action potentials, this acted as amplitude detector for action potentials within a certain frequency range. The degree of averaging of the amplitudes was varied by changing the size of the time constant. The two signals, representing respectively the positive and the negative phase of the original signal, were then led to a summation step. Between this and the output stage there was inserted a low-pass filter (L–C, constant K) with a suitable cut-off frequency at about 10 cycles per second. The output current from the filter, representing the mean amplitude of the input signal, was then led to a driver stage. To this were coupled two light-spot galvanometers, one built into the electro-

*) Designed by O. Sten-Knudsen, M.D.

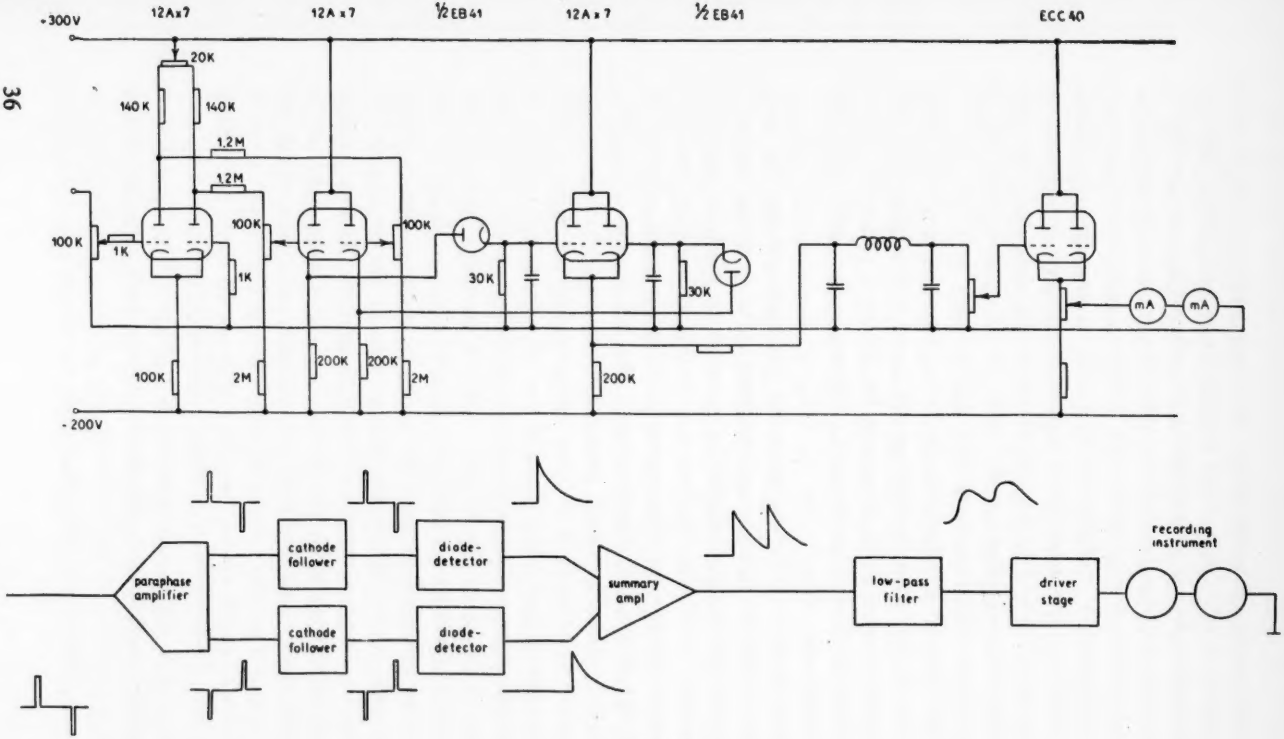

Fig. 11

Electronic device which gives an output current proportional to the mean amplitude of the action potentials. (*O. Sten-Knudsen*). Above: circuit diagram. Below: block diagram with schematic indication of the change in the shape of the action potentials as they pass through the different

myograph and the other in parallel for observation. The simultaneity with the oscillographically recorded deflections was ascertained. This apparatus was only used for measurement under relatively constant conditions and not during the transition from rest to contraction.

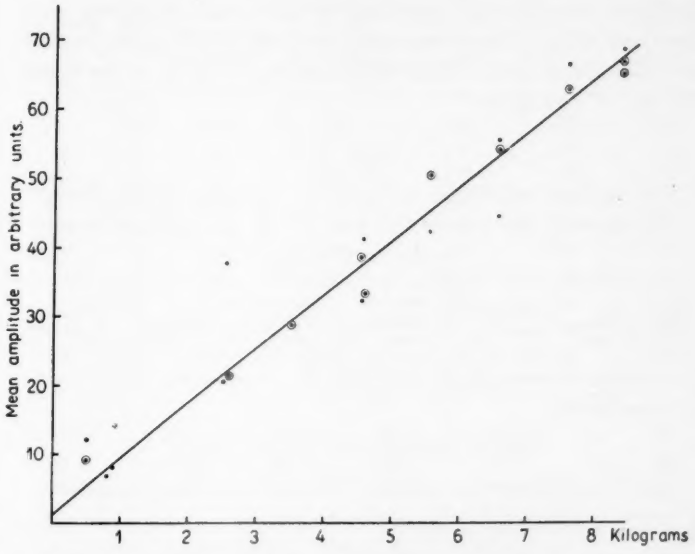

Fig. 12

Mean amplitude of the action potential pattern in arbitrary units as a function of the weight in kilograms with approximately isometric contractions of the brachial biceps muscle in two subjects ⊙ and · . The straight line relationship between the degree of muscle activity and the mean action potential amplitude is indicated by the line.

The correlation between the mean amplitude and the degree of activity was investigated in the brachial biceps of two subjects. The arm and wrist were fixed on an arm board with the arm flexed 90° at the elbow. The strength of isometric muscle contraction was varied by varying a weight attached to the hand. The action potential pattern was recorded with the concentric needle electrode described on p. 34. The maximum action potential amplitude varied about proportionally with the muscle force (Fig. 12).

Recording from two different places in the same intrinsic laryngeal

Electronic device which gives an output current proportional to the mean amplitude of the action potentials. (*O. Sten-Knudsen*). Above: circuit diagram. Below: block diagram with schematic indication of the change in the shape of the action potentials as they pass through the different

muscle under the same experimental conditions and with maximal muscle activity (high tones in the chest register), different maximal amplitudes were obtained. (These were no reflection of changes in amplitude of the single motor unit potential whose amplitude does not vary with the degree of activity). A large variation in maximal amplitude was therefore necessary to allow any conclusion as to significant differences as exemplified in Table 2, where the maximal amplitude found in the vocal muscle during phonation of the vowels "a" and "e" are given.

Table 2

The maximal amplitude of the action potential pattern during phonation of the vowels "a" and "e" at two different points in the vocal muscle

Experiment number	Phonation	Phonation frequency in c. p. s.	Maximal amplitude lead I in μV	Maximal amplitude lead II in μV	Difference in μV	Difference in per cent of lead I
1) Normal larynx.						
44	e	345	224	>580	>356	>159
44	a	222	322	>536	>214	> 66
44	e	285	358	>568	>210	> 59
2) Normal half of paretic larynx.						
37	e	(284) [1]	>870	673	>197	> 23
37	a	(262)	>870	540	>330	> 38
37	e	(294)	750	400	350	47
40	a	224	431	388	43	10
40	e	345	>435	>435	> 0	> 0
40	a	250	229	214	15	7
40	e	400	229	384	155	68
Mean value			472 (S. D. 40 per cent)	472		

[1]) Phonation frequencies in parantheses indicate experiments in which the phonation frequency was measured in immediately preceding or following phonations.

A similar difference in the amplitude of the action potential pattern was found in symmetrical muscles. The maximal amplitudes recorded simultaneously from the right and left cricothyroid muscles during maximal effort in subjects with normal larynx are given in Table 3.

Table 3

The maximal amplitude of the action potential pattern during phonation in the right and left cricothyroid muscle in subjects with normal larynx

Experiment number	Phonation	Phonation frequency in c. p. s.	Maximal amplitude in the right cricothyroid muscle in μV	Maximal amplitude in the left cricothyroid muscle in μV	Difference in μV	Difference in per cent of right muscle
12	e	267	188	486	298	158
12	a	167	425	400	25	6
12	e	358	900	570	330	37
12	a	167	350	360	10	3
12	e	358	650	560	90	14
12	e	143	160	275	115	72
12	e	286	295	345	50	17
12	e	500	195	280	85	44
44	e	345	314	394	80	25
44	e	(345)	264	198	66	25

Statistical Treatment of the Results

The variation in the values for duration and amplitude around the mean value (sum of the single values divided by their number) is described by the standard deviation (S.D.), defined as

$$\text{S. D.} = \sqrt{\frac{\Sigma\, d^2}{N - 1}}$$

with d the deviation of the single values from the arithmetical mean value and N the number of values.

The arithmetical mean value is determined with an uncertainty, the mean error, S.E., given by

$$\text{S. E.} = \frac{\text{S. D.}}{\sqrt{N}}$$

From the mean error the statistical significance of differences in potential duration and amplitude could be determined from

$$\eta = \frac{t'_m - t_m}{\sqrt{\text{S. E.}'^2 + \text{S. E.}^2}}$$

where t'_m and t_m are the mean values whose significance was to be determined and S.E.′ and S.E. the corresponding mean errors.

If η is larger than 2.5 there is a more than 99 per cent probability that the difference is real. Whenever a difference in mean duration or amplitude is termed real, the value of η has been at least 2.5.

IV. SOURCES OF EXPERIMENTAL ERROR

1. *Artifacts*

Mechanical movements of the electrodes were the most serious source of artifact in the electromyographic recordings. Such movements caused slow regular deflections of the base line (Fig. 13). However, on single sweep recordings the content of fast components identified the action potentials. *Van den Berg* and *Spoor* (1957)

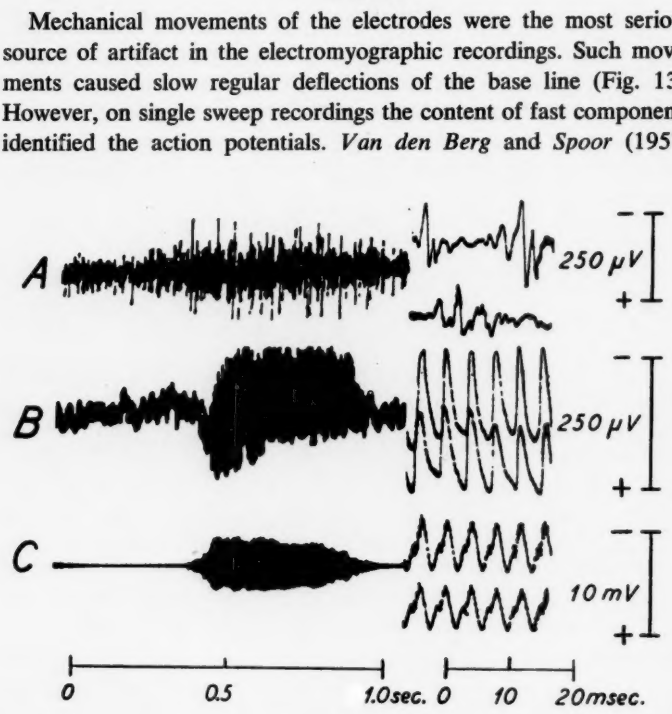

Fig. 13

Action potentials recorded continuously (left) and by single sweeps (right).
A. Action potentials in the left cricothyroid muscle.
B. Artifact from movement of the electrode in the left vocal muscle.
C. Microphone recording.
Patient no. 49, a 59 year old man with the left vocal cord immovable in the intermediate position.
Phonation: "e". Frequency 250 c.p.s.

consider these movement artifact potentials the explanation for a synchronism claimed between the potentials in the n. recurrens and the sound pattern (cf. p. 22, *Moulonget* 1955).

2. *Electrode Placement*

The electrode placement in the inner larynx was inspected before and after each experiment with a laryngeal mirror, in order to insure that the recorded potentials were from the muscle it was desired to investigate. In addition to the point of insertion in the muscle, the depth of insertion was observed, since with too deep insertion the tip of the needle electrode might pass through the muscle under investigation into another muscle. For example the needle electrode might traverse the thyro-arytenoid muscle and record from the lateral crico-arytenoid muscle or pass through the arytenoid muscle into the posterior crico-arytenoid muscle. However, when the thickness of the intrinsic muscle of the larynx is known (p. 43) this source of error is avoided.

3. *Microphone Distance*

To record comparable sound amplitudes with the microphone both in the individual experiment and from experiment to experiment, the distance between the subjects' mouth and the microphone was maintained constant. Were this distance not constant the amplitude of the microphonic deflection would be a source of error in comparing tone intensities.

4. *The Voice of the Subjects and of the Patients During the Investigation*

The electrodes used for investigation of the intrinsic laryngeal muscles weighed about four grams when hung over a support as they hung over the base of the mouth in an experiment. When such an electrode is inserted into a vocal cord it increases the mass of the muscle, thus influencing the vibration of the vocal cord and possibly the voice of the subject. However, a comparison of the voice during and after the investigation as recorded on the tape recorder demonstrated that there was no difference. Nor was there a difference in the voice of the same subject in experiments with an electrode

placed in the vocal cords and in experiments when no electrode was placed in the vocal cords. The same was found when the voice was compared in experiments with one electrode in the vocal cords and experiments with two electrodes in the vocal cords of the same subject. Nor did the local anesthesia influence the voice of the subjects. Mucus in the larynx was in certain instances not prevented by previous administration of atropin (p. 32) and could cause a change in the subjects' voice so that mucus had to be evacuated by coughing.

RESULTS

Anatomical Investigations

I. THE SIZE OF THE INTRINSIC LARYNGEAL MUSCLES

The intrinsic laryngeal muscles from two male and two female cadavers were removed and measured at that place where each muscle appeared to be about its average length, breadth and thickness (mean values in Table 4). The volume of the muscles was calculated from the mean length, breadth and thickness, and the weight by

Table 4

The size of the intricsic laryngeal muscles

Mean value of four excised human larynges (two men, weight 68 and 80 kg, height 172 and 180 cm; two females, weight 52 and 63 kg, height 160 and 165 cm)

	Length in mm	Breadth in mm	Thickness in mm	Cross sectional area in mm^2	Volume in mm^3	Calculated weight in mg	Weight in mg [1])
Cricothyroid muscle	15	10	6	60	900	954	940
Vocal muscle	15	4	5	20	300	318	357
Transverse arytenoid muscle	15	7	5	35	525	556	558
Posterior crico-arytenoid muscle	12	10	5	50	600	636	625

[1]) According to *Grabower* (1904).

multiplying the volume by the specific gravity 1.06 (Table 4). The last column of Table 4 shows *Grabower*'s (1904) measured weights

for the single muscles which are of about the same order of size as the calculated weights.

According to *Möller* and *Fischer* (1904) the distance between the anterior arch of the cricoid cartilage and the inferior edge of the thyroid cartilage was about 50 per cent less during phonation of high tones than at rest. I have measured the length of the vocal cords when this distance was reduced. With a reduction of the distance between the anterior arch of the cricoid cartilage and the inferior edge of the thyroid cartilage the membranous portion of the vocal cords was lengthened by about 30 per cent. This increase in length is in agreement with the 25 per cent lengthening found by X-ray measurements of the larynx during phonation at increasing pitch (*Sonninen* 1954, p. 13). Thus, this lengthening of the vocal cords is accounted for by the shortening of the cricothyroid muscle.

II. THE NUMBER OF MUSCLE FIBRES AND THE INNERVATION RATIO IN THE INTRINSIC LARYNGEAL MUSCLES

There is in the literature no report as to the mean number of muscle fibres in the intrinsic laryngeal muscles but the thickness of the individual muscle fibres has been determined (*Schwalbe* and *Mayeda* 1890) (Table 5). Utilizing this determination and the mean value for the thickness and breadth of the different intrinsic laryngeal muscles, the number of muscle fibres can be calculated as

$$\frac{\text{muscle breadth} \times \text{muscle thickness}}{\pi \times \left(\frac{\text{fibre diameter}}{2}\right)^2} = \text{number of muscle fibres}$$

In this calculation the size of the interstitial space must be corrected for by an average of 9.6 per cent (*Boyle, Conway, Kane* and *O'Reilly* 1941). The number of muscle fibres in the intrinsic laryngeal muscles according to this calculation is shown in Table 5.

A motor unit is the bunch of muscle fibres innervated by a single ganglion cell (*Sherrington* 1930). A count of the number of nerve fibres at their point of entrance into a muscle allows calculation of the number of motor units in the muscle if correction is made for

1. The number of sensory nerve fibres – 33 to 50 per cent – in human mixed nerves (*Sherrington* 1894, *Rexed* and *Thermann* 1948, *Fex* and *Wohlfart* 1954).

2. Double innervation of the same muscle fibre.

It is not known to what extent there may be double innervation of muscle fibres in the intrinsic laryngeal muscles (*Tiegs* 1953). Staining of end plates by *Koelle*'s cholinesterase stain has shown that the end plates in the cricothyroid muscles are localized to a strip, which covers about 15 per cent of the length of the muscle fibres (Fig. 14). In view of this finding a double innervation of any considerable number of muscle fibres in the intrinsic laryngeal muscles is hardly likely.

3. Nerve fibres supplying the intrafusal muscle fibres in the muscle spindles.

It has been claimed that muscle spindles do not exist in the intrinsic laryngeal muscles (*Sherrington* 1897, *Cilimbaris* 1910, *Rio-Hortega* 1925, *Fernand* and *Young* 1951). It is in agreement with the assumption that *Murray* (1957) found a unimodal distribution of fibre diameters in the inferior laryngeal nerve of the cat. However, absence of muscle spindles has also been claimed in the case of other muscles, such as the external ocular muscles, which were found on later investigation to be well supplied with muscle spindles (*Cooper* and *Daniel* 1949). Since muscle spindles have been demonstrated in the vocal muscle (*Goerttler* 1950) it seems most probable that muscle spindles are also present in the other intrinsic laryngeal muscles.

In the inferior laryngeal nerve of a 65 years old man without signs or symptoms of laryngeal paresis I have determined the distribution of fibre diameters two centimeters before the nerve's entrance into the muscle. The nerve was stained with a one per cent osmic acid solution and 4–5 μ thick sections were microphotographed and the diameters measured on photographic enlargements. The histogram of 1097 nerve fibre diameters is given in Fig. 14 A. Although there is no clear indication of a bimodal distribution, the great incidence of diameters below 10 μ suggests the presence of two groups of nerve fibres, one with its peak at 14 μ and another with the maximum incidence at 6–10 μ. Fibres of less than 10 μ diameter comprise 17 per cent of all fibres and these correspond probably to small nerve fibres supplying intrafusal muscle fibres of muscle spindles.

In Table 5 the number of motor units in certain of the intrinsic laryngeal muscles is calculated from *Grabower*'s (1904) count of nerve fibres with correction for the number of fibres to the intrafusal

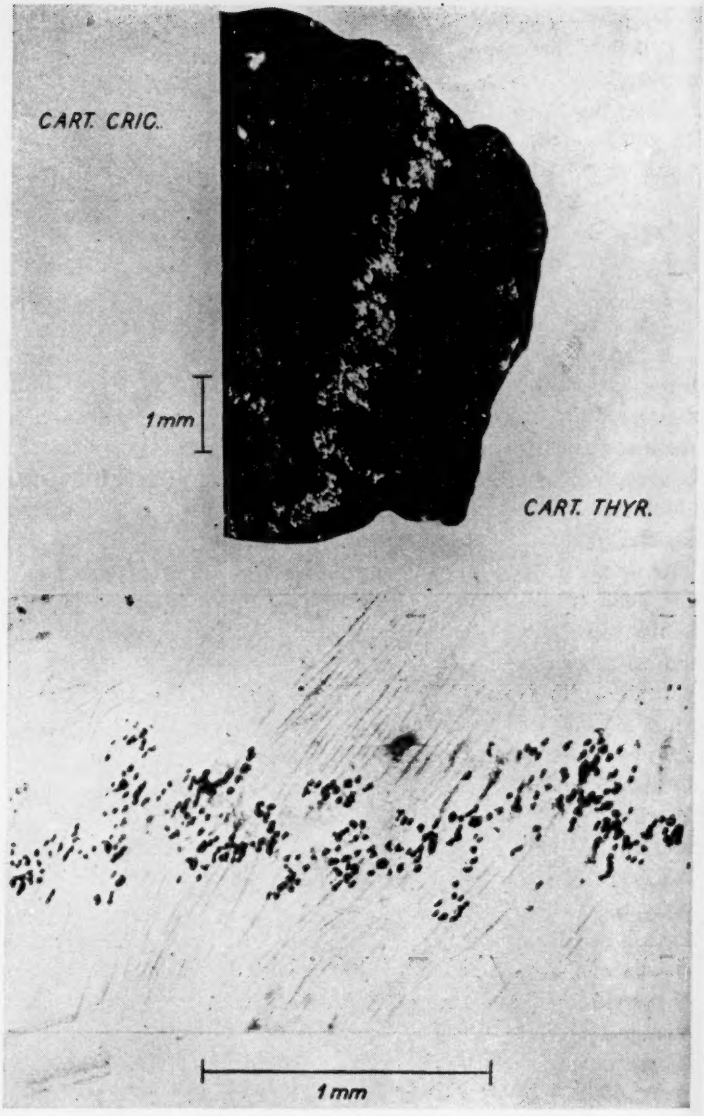

Fig. 14

The cricothyroid muscle from a child. End plate stain according to *Koelle*'s method (cholineesterase stain) *).

Above: end plates localized to a narrow band comprising about 15 per cent of the length of the muscle fibres.

Below: microphotograph of the end plates.

*) The author is indebted to Erna Christensen M.D. for the histological preparations.

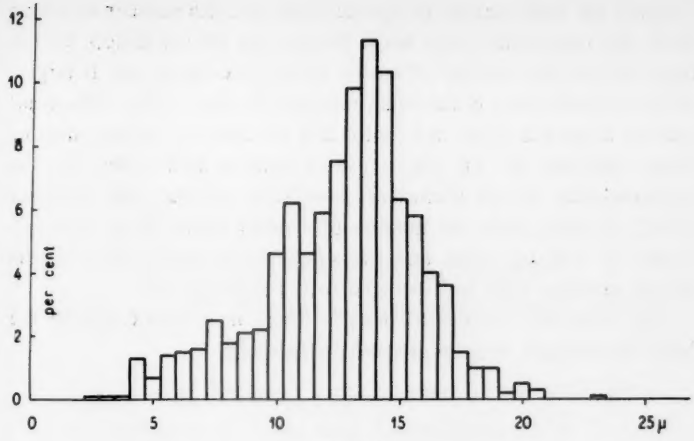

Fig. 14 a

Distribution of nerve fibre diameters in the inferior laryngeal nerve of a 65 year old man (1097 nerve fibres).
Ordinate: per cent of nerve fibres in cash diameter interval.
Abscissa: mean diameter in μ.

fibres in the muscle spindles (17 per cent) and of sensory fibres (33 per cent).

Table 5

Number of muscle fibres and innervation ratio for the intrinsic laryngeal muscles

Muscle	Muscle fibre diameter in mm[1]	Total number of muscle fibres[2]	Total number of nerve fibres[3]	Calculated number of motor units[4]	Number of muscle fibres per motor unit
Cricothyroid	0.0410	18550	225	112	166
Transverse arytenoid	0.0225	34470	279	139	247
Posterior crico-arytenoid	0.0388	16200	281	140	116

[1]) *Schwalbe* and *Mayeda* (1890).
[2]) Calculated from the cross sectional areas given in Table 4 corrected for 9.6 per cent interstitial space and the diameters given in column 1.
[3]) *Grabower* (1904).
[4]) Correcting for 33 per cent sensory fibres and 17 per cent efferent fibres to intrafusal fibres of muscle spindles.

Given the total number of muscle fibres and the number of motor units, the innervation ratio for a muscle can be calculated. In the large muscles the number of muscle fibres in a motor unit is larger, on the average, than in the small muscles (*Tergast* 1873). The mean number of muscle fibres in a motor unit for different human muscles varies between 25 for the platysma muscle and 1640 for the gastrocnemius muscle (*Feinstein, Lindegård, Nyman* and *Wohlfart* 1954). In their study the number of sensory nerve fibres was corrected for (40 per cent) and the small motor nerve fibres to the muscle spindles were not included in the calculations.

The calculated number of muscle fibres in a motor unit in the internal laryngeal muscles is given in Table 5.

Electromyographic Findings in Subjects with Normally Movable Vocal Cords and in Patients with Unilateral Vocal Cord Paresis in whom the Normal Side was Investigated.*)

I. SINGLE MOTOR UNIT POTENTIALS

The individual motor unit potentials recorded from the intrinsic laryngeal muscles were di- or triphasic. The amplitude was 100–300 μV and the duration 3–6 msec (Fig. 15). The amplitude and duration were of the same size order as in the external ocular muscles and in the muscles innervated by the facial nerve. The other skeletal muscles have a duration 50–80 per cent longer (*Buchthal* and *Rosenfalck* 1955). The shorter duration of the motor unit potentials for the intrinsic laryngeal muscles in comparison with the other skeletal muscles is related to the low "innervation ratio" (p. 47). Table 6 shows the mean action potential duration in various intrinsic laryngeal muscles.

Table 6

Mean action potential duration in various intrinsic laryngeal muscles

The mean age of the subject in the four groups was between 50 and 56 years, and no correction was made for age

Cricothyroid muscle	5,3 $\pm$ 0,1 msec (201 different potentials)
Vocal muscle	3,5 $\pm$ 0,1 msec (157 different potentials)
Arytenoid muscle	4,6 $\pm$ 0,2 msec (55 different potentials)
Posterior crico-arytenoid muscle	4,4 $\pm$ 0,2 msec (43 different potentials)

*) Preliminary report: *Faaborg-Andersen* and *Buchthal* 1956.

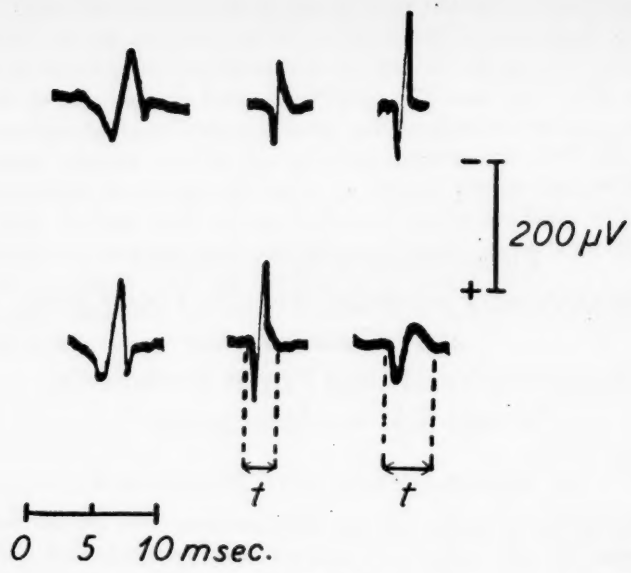

Fig. 15

Examples of motor unit potentials in the cricothyroid muscle, the vocal muscle and the thyro-arytenoid muscle. The measured action potential duration (t) is indicated by the stippled lines.

II. THE ACTION POTENTIAL PATTERN IN THE ABSENCE OF PHONATION

In the normal larynx and on the normal side in patients with unilateral paresis of the vocal cords, there was found electrical activity in the intrinsic laryngeal muscles during quiet respiration in spite of every effort to achieve relaxation. Even when the subjects held their breath this activity was observed. This electrical activity was of low amplitude and the single motor unit potentials could not be distinguished from each other. The electrical activity recorded represents, therefore, discharges from muscle fibres at a considerable distance from the recording electrode.

In the cricothyroid, vocal, thyro-arytenoid and arytenoid muscles the mean amplitude of this electrical activity was up to 200 μV, and varied considerably from subject to subject and in a single subject. In only 5 of the 32 normal subjects was there no electrical

activity in these muscles during quiet respiration. The amplitude of the resting electrical activity in the posterior crico-arytenoid muscle was higher, on the order of 200–500 μV, and was present in the absence of phonation in all subjects.

During quiet inspiration there was an increase in amplitude of the action potential pattern over and above its amplitude at rest of 20 –500 μV in the vocal muscle in all but one subject. In the other adductor muscles there was a corresponding increase in electrical activity during inspiration in about half the subjects. During expiration there was no change in electrical activity as compared with that found at rest (Fig. 16). In the posterior crico-arytenoid muscle,

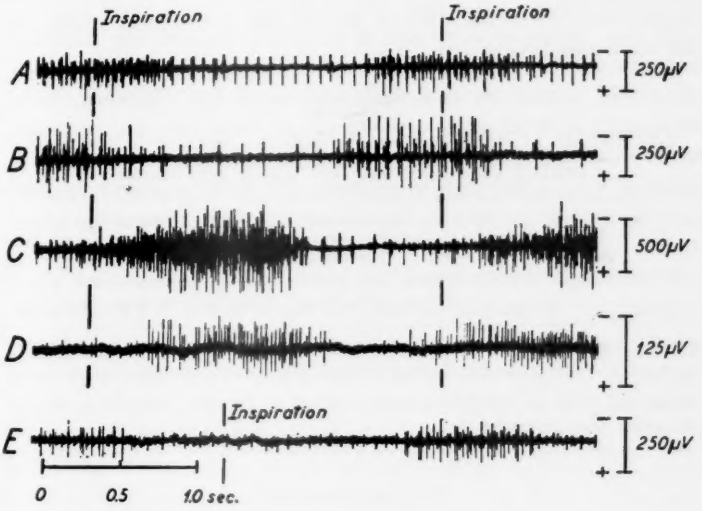

Fig. 16

Quiet respiration. The onset of inspiration is indicated by the vertical stippled lines.

A and B. Left cricothyroid muscle.
C and D. Left vocal muscle.
E. Left posterior crico-arytenoid muscle.
A and E: subject no. 29, a 54 year old woman with normally movable vocal cords.
B and C: subject no. 32, a 78 year old man with normally movable vocal cords.
D: subject no. 45, a 53 year old man with normally movable vocal cords.

in the majority of subjects, the amplitude of the action potential pattern was considerably reduced or all electrical activity disappeared during inspiration (Table 7), while during expiration the amplitude was 200–500 μV (Fig. 16).

Since under the experimental conditions it was difficult to signal the exact point of onset of respiration, even when recording from a thermocouple placed in the nose, the time relationships between inspiration and the change in electrical activity in the electromyogram could not be determined more accurately. A difference in time relationships between the muscles under investigation could be determined, and it was found that the increase in electrical activity in the arytenoid muscle began approximately 0.5 seconds before the increase in electrical activity in the cricothyroid muscle, which began about 0.9 seconds before the increase in electrical activity in the vocal muscle (Fig. 16).

Table 7

The number of subjects with and without increase in electrical activity during normal and forced respiration

Muscle	Ordinary respiration Change in potential pattern amplitude			Forced respiration Change in potential pattern amplitude	
	Increase	No change	Diminution	Increase	No change
Cricothyroid	9	5		7	2
Vocal	7	1		1	0
Arytenoid	3	2		2	1
Posterior crico-arytenoid		2	4	2	1

Forced Respiration

During forced respiration an increase in electrical activity during inspiration was found more frequently than during quiet respiration. In the cricothyroid, vocal and arytenoid muscles of the majority of subjects an increase occurred in the amplitude of the action potential pattern above that found at rest by 200–600 μV, but no change during expiration (Table 7). Changes in the posterior crico-arytenoid muscle during forced inspiration were in contrast to those during quiet respiration: in two subjects there occurred an increase in amplitude of the action potential pattern over and above that of the

resting electrical activity and no change in one subject (Table 7). During expiration there was no change in the electrical activity as compared with rest.

These findings are summarized in Table 7, where the number of subjects in whom the different muscles were investigated is shown, and the number with and without an increase in electrical activity during ordinary and forced respiration.

In summary, in the majority of subjects a slight degree of resting electrical activity was found in the adductor muscles. Since this activity did not disappear even when the subject ceased respiration it must be considered to be a "postural" electrical activity, presumably evoked through a proprioceptive reflex mechanism. During inspiration this electrical activity increased in amount and in amplitude, while it was not affected by expiration. During forced inspiration the increase in electrical activity was greater and more frequent than during quiet inspiration. In some subjects who had slight electrical activity at rest there was no change during inspiration. On the contrary, in the few subjects who showed no electrical activity at rest, an increase in electrical activity occurred during inspiration in all. The increase in electrical activity occurred first in the arytenoid muscle, 0.5 seconds later in the cricothyroid and 0.9 seconds after this in the vocal muscle.

In the abductor muscles there was a pronounced degree of electrical activity at rest in all subjects. During inspiration this electrical activity was inhibited in most subjects and it was unchanged during expiration. During forced inspiration the electrical activity increased in the abductor muscles in two of three subjects, in contrast to the findings during quiet respiration.

III. PHONATION

a) Electromyographic Findings During Phonation

The subjects were instructed to phonate a certain vowel with medium and constant intensity and of a constant pitch about the middle of the range of that particular subject's voice. During such phonation electrical activity in the cricothyroid, thyro-arytenoid, vocal and arytenoid muscles increased markedly. There was no systematic difference between the vocal and the thyro-arytenoid

muscles, either at rest or with phonation. This increase in electrical activity began and often reached its maximum before an audible tone was recorded by the microphone. At the point of onset of the audible tone as recorded by the microphone the electrical activity had often decreased slightly again but was still greater than in the absence of phonation. At the end of phonation, when audible tone had ceased as indicated by the microphone recording, the increase in electrical activity had also disappeared (Fig. 17). Occasionally,

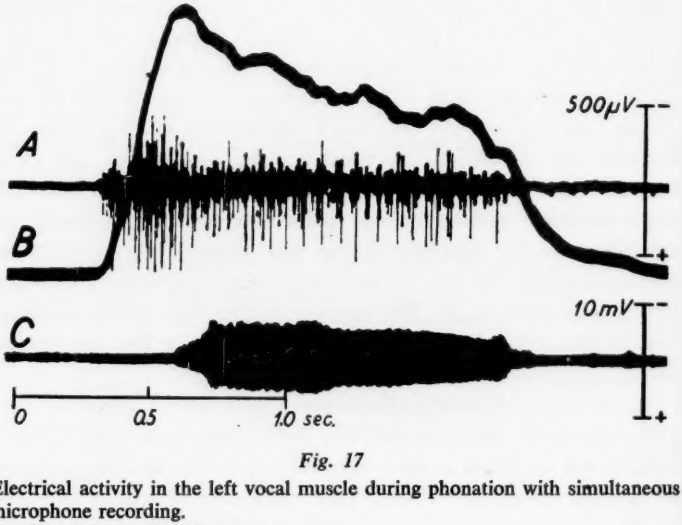

Fig. 17

Electrical activity in the left vocal muscle during phonation with simultaneous microphone recording.
A. The action potential pattern.
B. The mean action potential amplitude.
C. Microphone recording.
Phonation: "e". Frequency 285 c.p.s.
Patient no. 56, a 64 year old woman with the right vocal cord immovable in paramedian position. The *left* vocal cord was normally movable.

however, the increase in electrical activity disappeared somewhat after the audible tone.

Conditions were different in the posterior crico-arytenoid muscle. In this muscle there was a pronounced degree of electrical activity in the absence of phonation. With phonation this electrical activity was markedly inhibited (Fig. 18) from shortly before the onset of audible tone as indicated by the microphone recording throughout

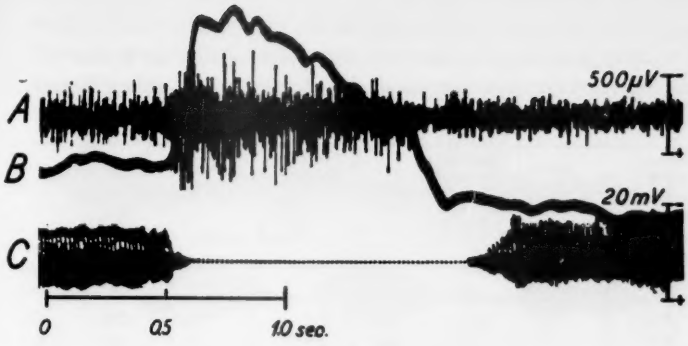

Fig. 18

Inhibition of activity in the posterior crico-arytenoid muscle during phonation.
A. The action potential pattern in the left posterior crico-arytenoid muscle.
B. Mean amplitude in the left posterior crico-arytenoid muscle.
C. Microphone recording.
Phonation: "a". Frequency 200 c.p.s.
Subject no. 57, a 47 year old man with normally movable vocal cords.

the whole period of phonation. In some instances, however, the inhibition decreased somewhat shortly after the onset of phonation.

The time interval ($\triangle$ t) between the start of the change in electrical activity and the onset of the tone as indicated by the microphone recording was 0.35 to 0.55 seconds. The variation was about 15 per cent for five to ten successive phonations repeated at short intervals as far as possible with the same intensity and frequency (Table 8). There was no significant difference between $\triangle$ t for the different muscles. There was no relationship between the tone pitch and $\triangle$ t, nor was there a relationship between the age or sex of the subject and $\triangle$ t.

The increase in electrical activity in the adductor muscles occurred nearly simultaneously and was accompanied by a reduction in electrical activity in the posterior crico-arytenoid muscle. Contrariwise, shortly before the cessation of phonation there occurred an increase in electrical activity in the posterior crico-arytenoid muscle and with cessation of phonation the electrical activity in the adductor muscles decreased. These changes in electrical activity occurred symmetrically on the two sides in normal subjects.

Thus the posterior crico-arytenoid muscle is an antagonist of the other intrinsic laryngeal muscles investigated in this study in that

Table 8

The time interval ($\triangle t$) between the onset of tone as indicated by the microphone recording and the change in the action potential pattern with phonation in subjects with normal larynx

Experiment number	Sex	Age in years	Phonation frequency in c. p. s.	$\triangle t$ msec	Experiment number	Sex	Age in years	Phonation frequency in c. p. s.	$\triangle t$ msec
Cricothyroid muscle					Vocal muscle				
21	♂	46	143	362	34	♂	63	(133)	390
46	♂	52	154	370	7	♂	70	167	344
7	♂	70	167	256	5	♂	45	200	348
27	♂	46	200	990	45	♂	52	200	400
29	♀	54	208	332	32	♂	77	208	697
32	♂	77	208	413	6	♀	46	250	480
31	♂	58	250	18	53	♂	52	(285)	297
13	♂	64	333	384	13	♂	64	333	386
7	♂	70	334	333	7	♂	70	334	353
44	♂	61	345	216					
Arytenoid muscle					Posterior crico-arytenoid muscle				
8	♂	54	200	700	27	♂	46	200	377
26	♂	77	238	550	29	♀	54	208	407
45	♂	52	250	666	31	♂	58	250	287
13	♂	64	250	140	17	♀	32	286	365
27	♂	46	286	333	17	♀	32	333	300
29	♀	54	333	733	30	♂	47	333	310
9	♀	48	435	700	17	♀	32	400	375
					16	♂	63	?	283

this muscle has a reciprocal innervation with respect to the other muscles. The vocal and arytenoid muscles are pseudo-antagonists as are the cricothyroid and arytenoid muscles (*Fulton* 1949), since they in fact function as synergists and are not connected by reciprocal innervation. The vocal and cricothyroid muscles are true synergists.

Although this study does not pretend to contribute to purely phonetic problems, there were certain experiments as to action potential amplitude during phonation of different vowels and consonants. A series of subjects were instructed to phonate different vowels, as for example "a-o-u-e" in immediate succession and as far as possible at the same intensity and pitch.

The action potential amplitudes were of the same order size of

all the vowels investigated in the cricothyroid, vocal, thyro-arytenoid and the arytenoid muscles. The same was true for the time relationships and for the interrelationships between the different

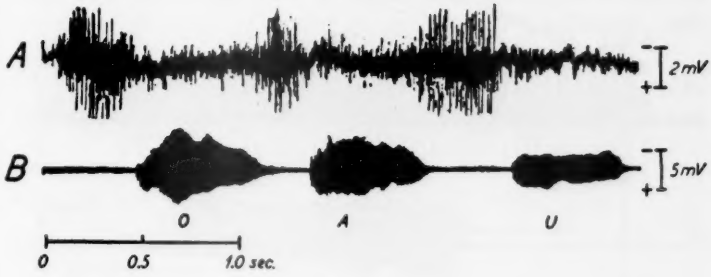

Fig. 19

Phonation of the vowels "o – a – u".
Frequency 200 c.p.s., the same for all three vowels.
A. The action potential pattern in the right thyro-arytenoid muscle.
B. Microphone recording which shows that the tone volume was somewhat less for "u" than for the two other vowels.
Subject no. 5, a 45 year old man with normally movable vocal cords.

muscles. In Fig. 19 is illustrated the action potential recording during phonation of the vowels "o-a-u".

In addition the electrical activity was recorded during phonation of single consonants. The potential pattern, for example, for the consonant "p" was very irregular, often consisting of repeated episodes of increase in electrical avtivity interrupted by short periods of almost no electrical activity. Since the phonation of vowels resulted in constant and reproducible potential patterns, the following investigations were in the main limited to phonation of vowels.

b) Discharge Frequency of the Single Motor Units

In six subjects with normal larynx it was possible during phonation to follow the frequency of a single motor unit without interference of activity from neighbouring units. In patients with paresis of the vocal cord it was easier to follow the discharge frequency of a single motor unit.

In the cricothyroid muscle (Fig. 20 a) the frequency (Fig. 20 b) increased in these experiments by up to 25 per second before the onset of audible tone. In the beginning of phonation the frequency

remained constant at about 15–20 per second, and fell again towards the end of phonation to 10–15 per second. Simultaneous with the cessation of phonation the action potentials ceased. The volume of the tone as recorded by the microphone decreased throughout phonation.

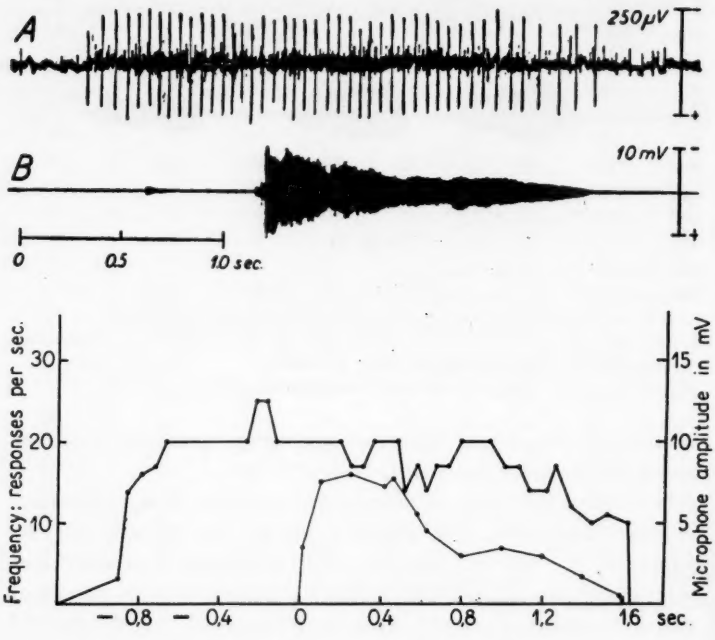

Fig. 20

a) Above: action potentials of a single motor unit during phonation.
   A. Right cricothyroid muscle.
   B. Microphone recording.

b) Below: discharge frequency for the single motor unit from the record above and the amplitude of the microphone recording in mV (thin line) as a function of time in seconds.

Phonation: "e". Frequency 286 c.p.s.

Patient no. 54, a 70 year old woman with the left vocal cord immovable in paramedian position. The *right* vocal cord was normally movable.

A similar increase in frequency was found in the vocal muscle. Fig. 21 a shows action potentials from one or from very few motor units in this muscle during phonation. Considering only potentials

of equal amplitude the discharge frequency of single motor units could be followed before, during and after phonation in three ex-

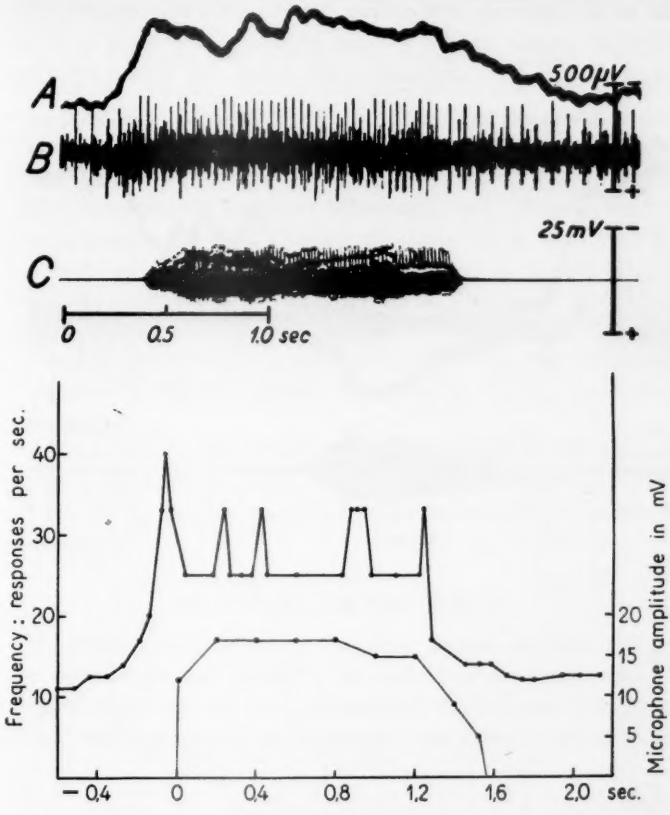

Fig. 21

a) Above: action potentials from one or very few motor units during phonation.
   A. Mean amplitude in the left vocal muscle.
   B. Action potential pattern in which the activity of one motor unit can be distinguished in the left vocal muscle by means of approximately equal amplitude for a series of discharges.
   C. Microphone recording.

b) Below: discharge frequency for the single motor unit from the record above and the amplitude of the microphone recording in mV (thin line) as a function of time in seconds.

Phonation: "e". Frequency 357 c.p.s.

Patient no. 57, a 47 year old man with normally movable vocal cords.

periments (one is illustrated in Fig. 21 b). In these experiments the frequency rose by from 10–40 per second before the onset of audible tone. In the beginning of phonation the frequency was constant about 25–30 per second and fell towards the end of phonation to 15 per second. Shortly after cessation of phonation, the resting frequency

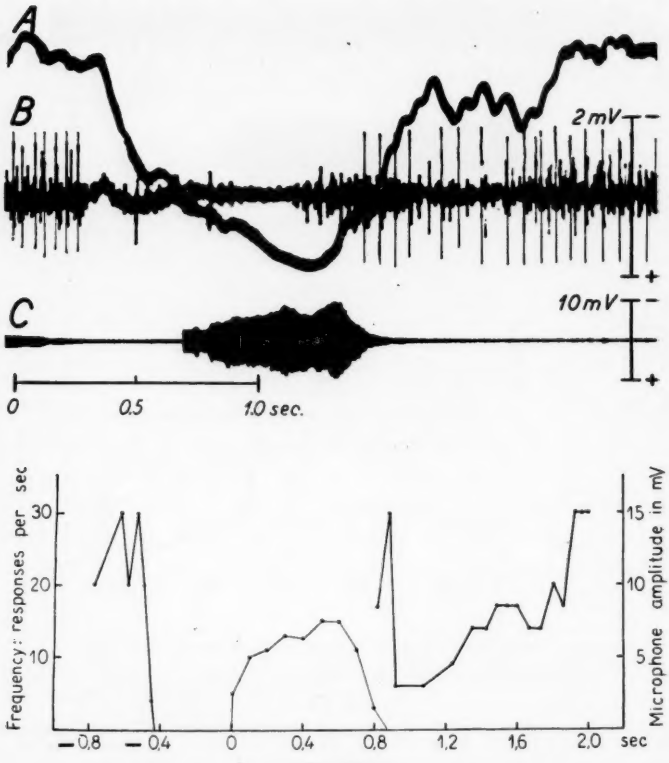

Fig. 22

a) Above: action potentials of a single motor unit during phonation.
   A. Mean amplitude in the right posterior crico-arytenoid muscle.
   B. Action potential pattern in the right posterior crico-arytenoid muscle.
   C. Microphone recording.

b) Below: discharge frequency for the single motor unit and the amplitude of the microphone recording in mV (thin line) as a function of time in seconds.

Phonation: "e". Frequency 333 c.p.s.

Subject no. 51, a 78 year old woman with normally movable vocal cords.

was about 12 per second. The intensity of tone, as shown by the microphone recording, was approximately constant, though it fell slightly at the end of phonation (Fig. 21 b).

In the arytenoid muscle it was not possible to obtain recordings showing the frequency change in a single motor unit. This was, however, possible in five experiments recording from the posterior cricoarytenoid muscle (Figs. 22 a and b), and in these experiments the frequency before phonation was 20–30 per second; 0.4–0.5 seconds before phonation the frequency fell abruptly to 5–10 per second and ceased entirely thereafter. During phonation there were no action potentials. A few milliseconds before the end of audible tone the frequency increased for a short period to 25–30 per second, and fell again to 5–10 per second and there after rose gradually to 20–25 per second in the course of about one second. The intensity of tone as recorded by the microphone was somewhat variable during these experiments.

As indicated by the above description and the illustrations, the discharge frequency for a single motor unit is maximally about 20–30 per second. In a single instance a frequency of 50 per second was recorded.

c) Alteration in Tone Volume

To investigate the effect of variations in tone intensity the subjects were instructed to say "e" in crescendo and decrescendo at constant pitch (Fig. 23). The amplitude of the envelope of the microphone recording and of the action potential pattern was measured

Fig. 23

Phonation with increasing tone volume.
A. The action potential pattern in the right thyro-arytenoid muscle.
B. Microphone recording.
Phonation: "e". Frequency 357 c.p.s. constant during the whole phonation.
Patient no. 9, a 45 year old woman with normally movable vocal cords.

at intervals of 100 msec. Neither the amplitude of the action potential pattern nor the electronically averaged amplitude varied as a function of the amplitude of the microphone recording (Fig. 24,

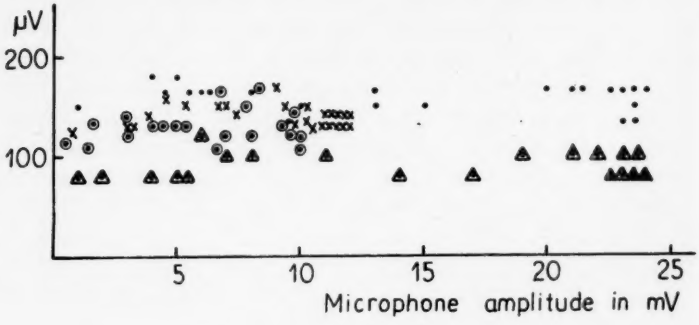

Fig. 24

The amplitude of the action potential pattern in the cricothyroid muscle in μV as a function of the amplitude of the microphone recording in mV for four subjects with normally movable vocal cords during phonation of the vowel "e" with increasing tone volume.

•	subject	no.	7	frequency	168	c.p.s.	69	year	old	man
△	–	–	43	–	200	–	33	–	–	–
⊙	–	–	27	–	286	–	46	–	–	–
×	–	–	9	–	357	–	45	–	–	woman

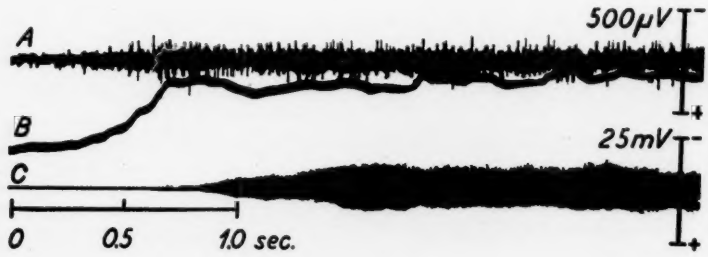

Fig. 25

Phonation with increasing tone volume.
A. The action potential pattern in the left vocal muscle.
B. Mean amplitude in the left vocal muscle.
C. Microphone recording.
Phonation: "e". Frequency 353 c.p.s. constant during the whole phonation. Subject no. 53, a 19 year old man with normally movable vocal cords.

Table 9), indicating that electrical activity did not increase in the intrinsic laryngeal muscles investigated when the intensity of tone increased (Figs. 25 and 26).

Table 9

The amplitude of the action potential pattern in μV with microphone amplitude of 2 and 10 mV

Reproduction of the tone on the tape recorder indicated that the frequency in some instances varied by 15–65 cycles per sec. for a given phonation

Experiment number	Phonation frequency in c. p. s.	Amplitude of the action potential pattern in μV for:					
		cricothyroid muscle with microphone amplitude of:		vocal muscle with microphone amplitude of:		arytenoid muscle with microphone amplitude of:	
		2 mV	10 mV	2 mV	10 mV	2 mV	10 mV
Subjects with normal larynx.							
7	(250)	165	165	170	175		
8	143	175	175	180	180		
9	285	300	300			150	130
9	357	140	140	210	220		
11	200	160	160	210	210		
13	(333)	500	525				
27	200	90	115			80	80
27	286	125	125				
29	333	105	125			100	125
30	263	210	210				
34	(133)	125	110	340	325		
43	200	85	85	520	540		
45	250			25	25		
The normal side of patients with unilateral vocal cord paresis.							
22	(250)	650	700	200	200		
24	263	70	80	350	390		
25	(200)	>680	>680	950	900		
28	222	400	350			170	115

d) Alteration in Tone Pitch

To investigate the effect of changes in pitch the subjects were instructed to phonate two vowels at different pitch in rapid glissando succession (as for example "a–he"). Every attempt was made in these experiments to ensure that the intensity of tone was constant. This was not possible in all instances, as shown by the amplitude

of the microphone recording, in that in some instances the intensity was greater with high than with low tone. As demonstrated in the preceding section (c) this difference in intensity could, however, not be expected to influence the amplitude of the action potential pattern. In the subjects with normal larynx there was in addition investigated the effect of change in pitch within the same register as well as

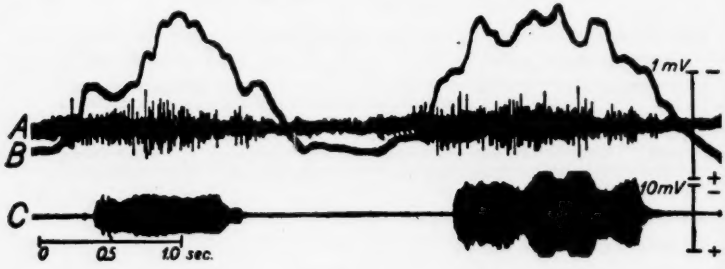

Fig. 26

Two immediately successive phonations with different tone volume.
A. The action potential pattern in the right vocal muscle.
B. Mean action potential amplitude in the right vocal muscle.
C. Microphone recording.
Phonation: "e". Frequency 285 c.p.s. for both phonations.
Subject no. 50, a 55 year old woman with freely movable vocal cords.

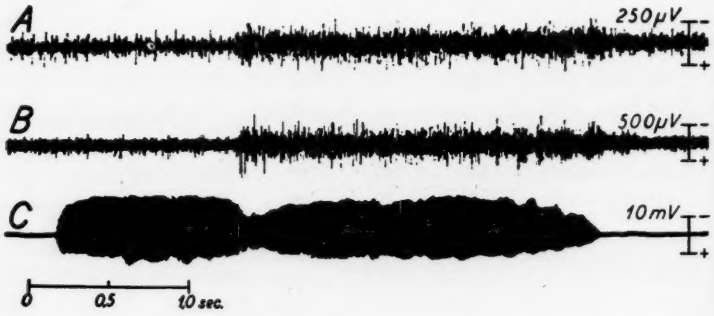

Fig. 27

Phonation at different pitch.
A. Action potential pattern in the left cricothyroid muscle.
B. Action potential pattern in the right cricothyroid muscle.
C. Microphone recording.
Phonation: "ahe". Frequency 167 c.p.s. for "a" and 358 c.p.s. for "e".
Subject no. 12, a 36 year old man with normally movable vocal cords.

changes in pitch with simultaneous change in register. The presence or absence of change in register was determined by listening to the phonations as recorded on the tape recorder. In the patients with unilateral paresis of the vocal cords it was impossible to determine whether there had been a shift in register during phonation. For this

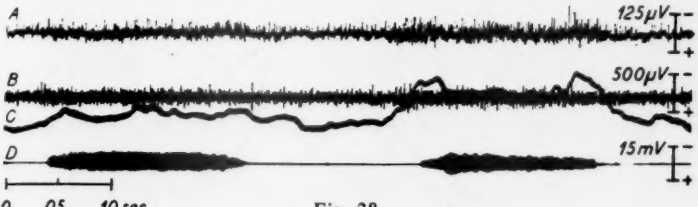

Fig. 28

Phonation at different pitch.
A. Action potential pattern in the left cricothyroid muscle.
B. Action potential pattern in the left vocal muscle.
C. Mean action potential amplitude in the left cricothyroid muscle.
D. Microphone recording.
Phonation: "a". Frequency 166 c.p.s. with the first phonation, 337 c.p.s. with the second phonation.
Subject no. 57, a 47 year old man with normally movable vocal cords.

reason investigations on the normal side of patients with unilateral vocal cord paresis could not be classified according to whether or not there was a change in register.

In normal subjects when the increase in the pitch took place within the same register there was an increase in the maximum amplitude of the action potential pattern by 200 to 600 per cent in all muscles investigated except in the arytenoid muscle where the increase in amplitude amounted to about 100 per cent. When the increase in pitch occurred with a simultaneous shift in register the changes in action potential pattern amplitude were variable. In the cricothyroid muscle there was an increase of about 100 per cent, in the vocal muscle no or only a small increase, and in the arytenoid muscle in some instances no increase and in some instances a marked increase in the amplitude of the action potential pattern (Figs. 27, 28, 29 and 30).

The amplitude change with pitch was less constant in patients with unilateral vocal cord paresis in whom the normal side was investigated than in normals, whether or not because of a shift in register could not be determined.

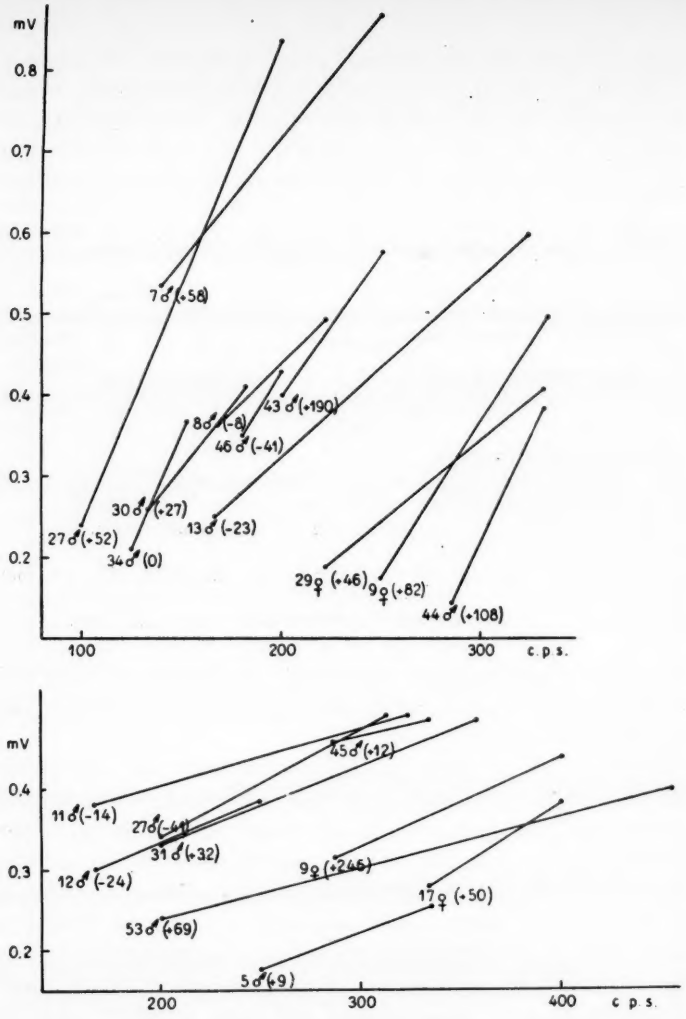

Fig. 29

Cricothyroid muscle.
Subjects with normally movable vocal cords. The maximal amplitude in mV of the action potential pattern as a function of the tone frequency in cycles per second for the cricothyroid muscle in experiments without change in register (above) and with change in register (below).
The numbers indicate the experiment number, the symbol the subject's sex and the numbers in parentheses the per cent change in tone volume from the low to the high frequency.

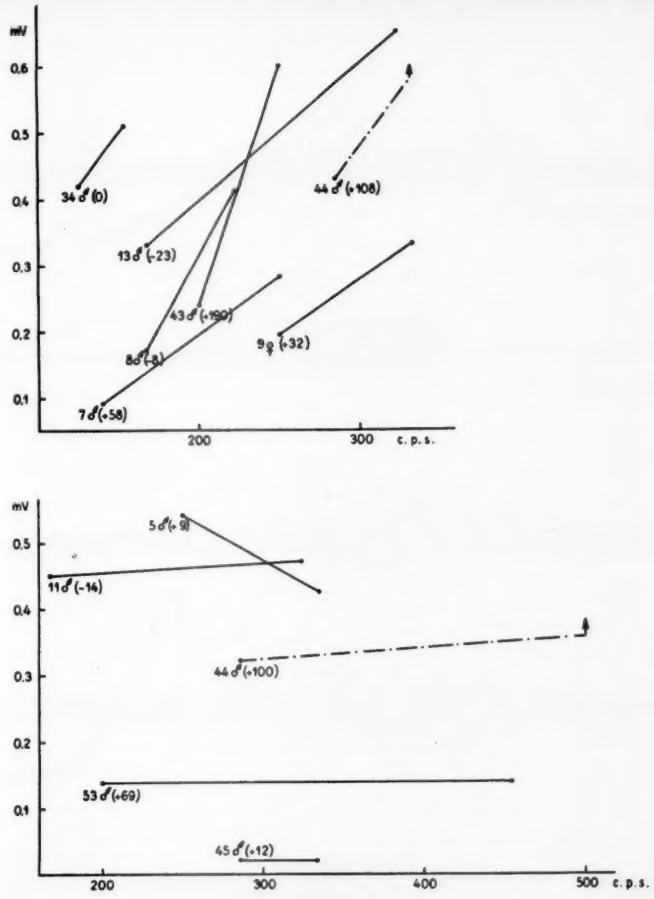

Fig. 30

Vocal muscle.
Subjects with normally movable vocal cords. The maximal amplitude in mV of the action potential pattern as a function of the tone frequency in cycles per second for the vocal muscle in experiments without change in register (above) and with change in register (below). The numbers indicate the experiment number, the symbol the subject's sex and the number in parentheses the per cent change in tone volume from the low to the high frequency. The stippled line represents an experiment in which it was only possible to determine the maximal amplitude for the high frequency as being greater than the value indicated by the arrow.

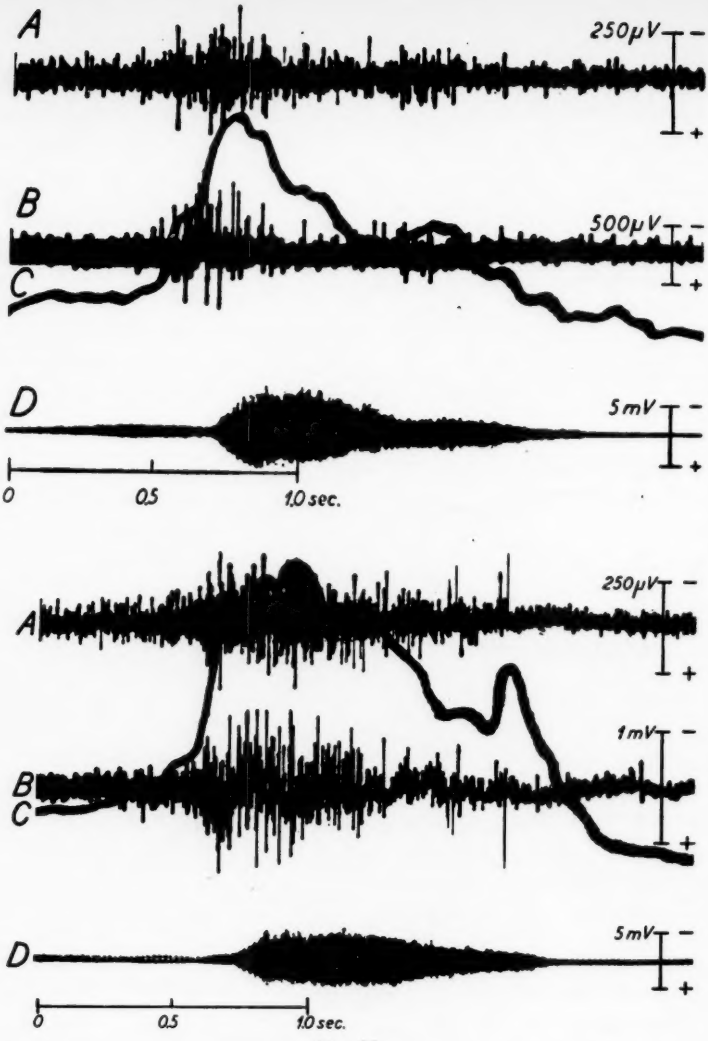

Fig. 31

Amplitude of the action potential pattern before and during phonation with different pitch in immediate succession.

A. Action potential pattern in the right cricothyroid muscle.

B. Action potential pattern in the right vocal muscle.

C. Mean action potential amplitude in the right cricothyroid muscle.

D. Microphone recording.

Phonation: "e". Frequency: above, 250 c.p.s.; belov, 355 c.p.s.

Patient no. 58, a 67 year old man with left vocal cord immovable in paramedian position. The right vocal cord was normally movable.

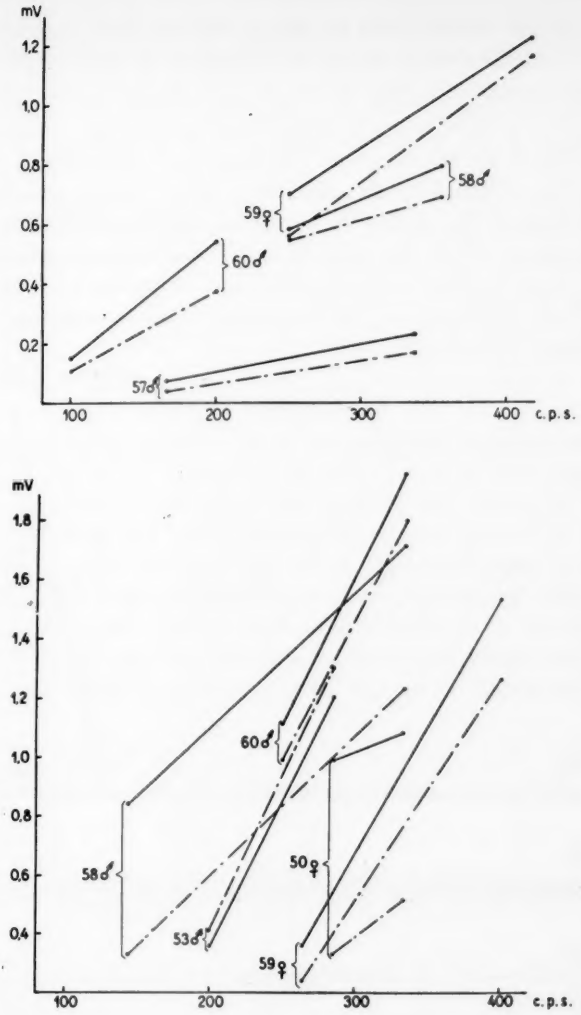

Fig. 32

The maximum amplitudes of the action potential pattern in mV before (stippled lines) and during (solid lines) phonation as a function of tone frequency in cycles per second for *the cricothyroid muscle* (above) and for *the vocal muscle* (below) in subjects with normally movable vocal cords (nos. 50, 53, 57 and 59) and in patients with unilateral vocal cord paresis where the normal side was investigated (nos. 58 and 60). The figures beside the lines indicate the experiment number, the symbols indicate the subject's sex.

e) The Amplitude of the Action Potential Pattern Before and During Phonation as a Function of Tone Pitch

As has been discussed in the preceding section the amplitude of the action potential pattern increased during phonation at increasing pitch within the same register. This increase occurred before audible tone was recorded by the microphone (Fig. 31); the increase was very close to that during the phonation itself (Fig. 32). Even with a change in register the electrical activity rose *before* a given phonation to nearly the level it had *during* it. This applied both when the amplitude of the action potential pattern was measured directly and averaged electronically.

f) Whispered Voice

To investigate the amplitude of the action potential pattern with whisper, four normal subjects were instructed to whisper the vowel "e". In all four subjects there was an increase in electrical activity (Fig. 33) in the cricothyroid muscle, though less than during ordinary speech, immediately before and after the whisper (Fig. 34). Similarly in three subjects investigated the maximal action potential amplitude in the vocal muscle was less with whispered than with ordinary speech though not so much less as in the case of the cricothyroid muscle. In the one subject investigated the maximal ampli-

Fig. 33

Phonation with whispered voice.
A. Action potential pattern in the right cricothyroid muscle.
B. Action potential pattern in the right vocal muscle.
C. Microphone recording.
Phonation: "a" with whispered voice.
Subject no. 11, a 60 year old man with normally movable vocal cords.

tude in the arytenoid muscle was greater with whispered than with ordinary speech. The fact that electrical activity was less in whispered than in ordinary speech indicates a difference between whispered and ordinary speech not due to intensity alone; with changes in intensity the amplitude of the action potential pattern remained unaltered (cf. p. 63).

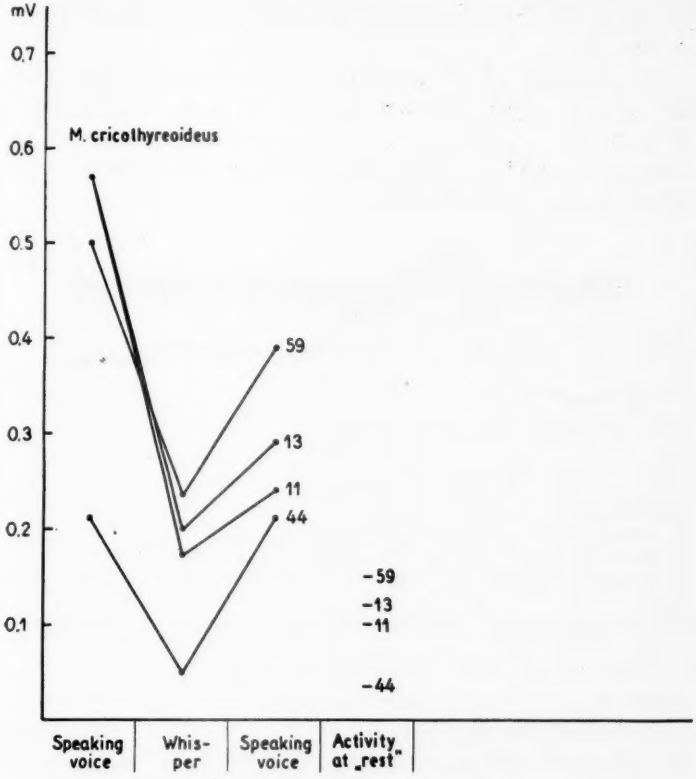

Fig. 34

The maximal amplitude of the action potential pattern in mV in the cricothyroid muscle in four subjects with normally movable vocal cords during three phonations in immediate succession, the first and the last with ordinary spoken voice and the middle with whispered voice. The figures beside the lines indicate the experiment number. The electrical activity at rest in each experiment is indicated to the right.

g) "Silent Speech"

Opinions differ as to the importance of "silent speech" in thinking. *Watson* (1924) suggested that thinking is always accompanied by movements in the intrinsic laryngeal muscles while *Wertheimer* (1945) considered "silent speech" to be of no importance in thinking processes. There are certainly large individual differences in this respect, as there are differences in the extent to which different people use that form of thinking which is called "talking to oneself" (*Miller* 1951).

The amplitude of the action potential pattern was investigated in a series of subjects who were instructed to think about phonation of the vowel "e" without audible phonation. Such a command is often accompanied by movements of the larynx of which the subject himself is not aware (Fig. 35).

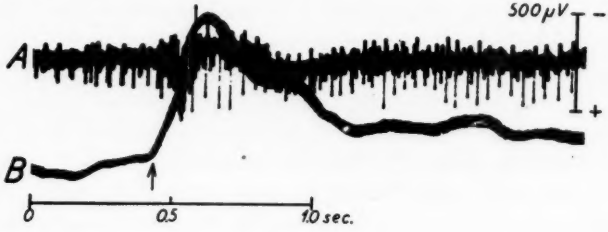

Fig. 35

"Silent speech".
A. Action potentials in the right vocal muscle.
B. Mean action potential amplitude in the right vocal muscle.
At the arrow the subject was instructed to think of saying "e" without audible sound.
Subject no. 59, a 28 year old woman with normally movable vocal cords.

"Silent speech" was accompanied by an increase in electrical activity and action potential amplitude in both the cricothyroid and the vocal muscles, but the increase was less than during immediately preceding and subsequent audible phonation (Fig. 36), markedly less in the cricothyroid muscle. The same changes were found on the normal side of three patients with unilateral vocal cord paresis.

h) Phonation with Different Types of Tone Initiation or "Attack"

By "attack" is understood the positioning of the vocal cords at the transition from rest to phonation. Three forms of attack can be

differentiated: "coup de glotte" or stopped attack ("op"), even attack or static initiation ("bob"), and breath attack ("hop"). For stopped attack the vocal cords are pressed against each other before the beginning of phonation. When the intra-tracheal pressure rises the vocal cords are forced apart at the moment of phonation. In the case of even attack the vocal cords are parallel at a distance of a few millimeters from each other before phonation. At the moment of phonation the vocal cords swing away from each other. In the case

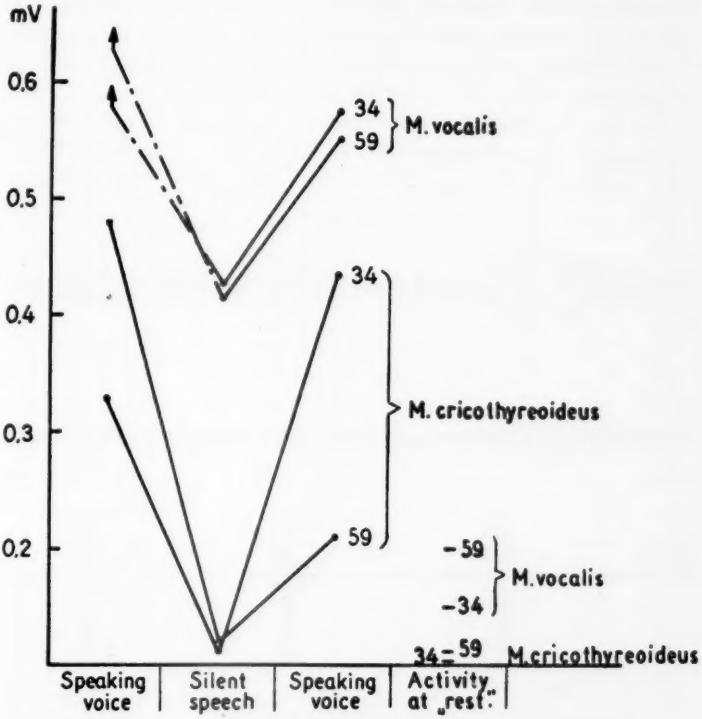

Fig. 36

"Silent speech".
The maximal amplitude of the action potential pattern in mV in the cricothyroid and vocal muscles in two subjects with normally movable vocal cords during three phonations in immediate succession, of which the first and the last were with spoken voice and the middle with "silent speech". The figures beside the lines indicate the experiment number. The electrical activity at rest in each experiments is indicated to the right.

of breath attack the vocal cords at the onset of phonation are in the same position as during respiration; from this position they immediately swing towards the midline and collide.

In order to investigate the action potential pattern during the different forms of attack, a series of subjects were instructed to repeat the syllables "op–bob–hop" sometimes in rapid and sometimes in slow succession as far as possible at the same intensity and pitch. In some experiments the order of the words was changed.

Table 10 shows the amplitude of the action potential pattern in the cricothyroid, vocal and arytenoid muscles, as well as the time interval between the onset of the increase in amplitude of the action potential pattern and the beginning of audible tone as indicated by the microphone record ($\triangle$ t) with the three forms of attack.

In most instances the amplitude of the action potential pattern was greater with phonation of "op" than with phonation of "bob" and "hop" (Fig. 37), specially in the vocal muscle. The difference

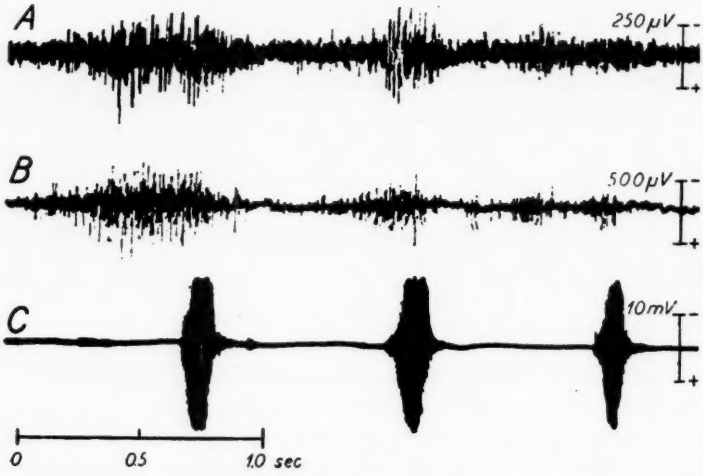

Fig. 37

Phonation with different attack.
A. The action potential pattern in the left cricothyroid muscle.
B. The action potential pattern in the left vocal muscle.
C. The microphone recording for "op" (stopped attack, furthest to the left), "bob" (even attack, in the middle) and "hop" (breath attack, to the far right).
Subject no. 13, a 65 year old man with normally movable vocal cords.

Table 10

The maximal amplitude of the action potential pattern, the microphone amplitude and the time interval between the onset of the change of the action potential pattern and the onset of tone as indicated by the microphone recording (△ t) in the cricothyroid, vocal and arytenoid muscles in subjects with normal larynx with different types of attack

Experiment number	Activity at rest in μV	Phonation: »OP«			Phonation: »BOP«			Phonation: »HOP«		
		△ t in msec	Microphone amplitude in mV	Potential pattern amplitude in μV	△ t in msec	Microphone amplitude in mV	Potential pattern amplitude in μV	△ t in msec	Microphone amplitude in mV	Potential pattern amplitude in μV
Cricothyroid muscle										
7	150	124	>28,1	436	140	28,5	480	126	27,4	373
8	120	196	13,4	280	160	14.5	260	150	14,0	320
8	120	163	12,8	306	149	12,9	272	145	11,7	236
8	120	187	26,3	366	173	25,2	314	166	24,3	300
9	100	198	18,4	294	164	15,7	199	152	22,5	370
11	150	135	>26,7	459	120	>26,1	456	108	21,2	414
11	150	125	25	356	117	24	330	125	24,7	290
13	100	303	28	530	137	26,3	460	135	22,1	420
13	100	145	10,5	548	130	11,5	558	137	11,0	484
20	60	200	23,8	344	130	21,5	370	134	22,8	404
32	200	195	26,6	574	144	25,9	526	160	28,4	576
9	100	178	22,1	514	142	24,1	526	135	25,9	454
34	150	?	18,6	292	?	15,2	292	?	23,2	382
Vocal muscle										
7	80	135	>28,1	352	133	>28,5	350	127	27,4	264
8	80	273	13,4	536	150	14,5	176	150	14,0	252
8	80	251	12,8	338	155	12,9	218	146	11,7	168
8	80	212	26,3	454	157	25,2	234	175	24,3	296
9	30	259	18,4	501	140	15,7	206	170	22,5	318
11	200	152	>26,7	429	140	>26,1	351	123	21,2	366
11	200	145	25	256	127	24	256	127	24,7	242
13	50	277	28	905	123	26,3	505	123	22,1	540
32	75	210	26,6	433	130	25,9	355	112	28,4	375
34	200	170	18,6	344	?	15,2	292	?	23,2	464
Arytenoid muscle										
9	60	193	22,1	360	152	24,1	345	145	25,9	246
13	100	132	10,5	294	137	11,5	288	132	11,0	250

in amplitude between "op" and "bob–hop" was significant in the case of the vocal muscle ("op" to "bob": mean difference 161 ± 30 μV, "op" to "hop": mean difference 126 ± 20 μV), but not in the case of the cricothyroid muscle. There was no significant difference in amplitude between "bob" and "hop".

The increase in amplitude of the action potential pattern occurred before the beginning of audible tone as recorded by the microphone in all subjects. The time interval ($\triangle$ t) was significantly greater with phonation of "op" than with phonation of "bob" and "hop". (The cricothyroid muscle: "op" to "bob": mean difference 37 ± 7 msec, "op" to "hop": mean difference 40 ± 8 msec. Vocal muscle: "op" to "bob": mean difference 68 ± 16 msec, "op" to "hop": mean difference 73 ± 14 msec). There was no significant difference in $\triangle$ t between "bob" and "hop".

In summarizing, in the vocal muscle phonation with stopped attack was associated with a greater increase in the amplitude of the action potential pattern than with even or breath attack and the increase in electrical activity occurred earlier. There was no significant difference between even and breath attack, nor was the difference between stopped, even and breath attack significant in the cricothyroid and arytenoid muscles.

i) Phonation With and Without Glottal Catch ("Stød")

The glottal catch is a peculiarity of the Danish language denoted "stød". It has been investigated partly phonetically by ear (*Høysgaard* 1747, *Storm* 1874, *Verner* 1881, *Thomsen* 1897, *Forchhammer* 1898) and partly by instrumentation (*Rousselot* 1924, *Selmer* 1925, *Ekblom* 1930, *Heger* 1931, *Smith* 1944). *Smith* found that the glottal catch ("stød") is chiefly produced by an increase in subglottal pressure while the contribution of the vocal cord is more uncertain.

In order to investigate the degree of electrical activity of the intrinsic laryngeal muscles during phonation with and without glottal catch, the amplitude of the action potential pattern was investigated when a series of subjects phonated alternately with and without glottal catch. The test words were "Mor" without glottal catch, and "Mord" with glottal catch; "Møller" without glottal catch, and "Møller" with glottal catch.

Fig. 38 shows an example of phonation of the word with and without glottal catch. In Table 11 are given the amplitude of the action potential pattern in the cricothyroid, vocal and arytenoid muscles as well as the time interval between the onset of the increase in amplitude of the action potential pattern and the beginning of audible tone as indicated by the microphone record (△ t) during phonation of words with and without glottal catch.

The increase in electrical activity in the vocal muscle was relatively greater with phonation of words with than of words without glottal catch. In the cricothyroid muscle there was on the other hand no difference. The time interval between the increase in amplitude of the action potential pattern and the beginning of audible tone as indicated by the microphone record (△ t) also showed no systematic difference.

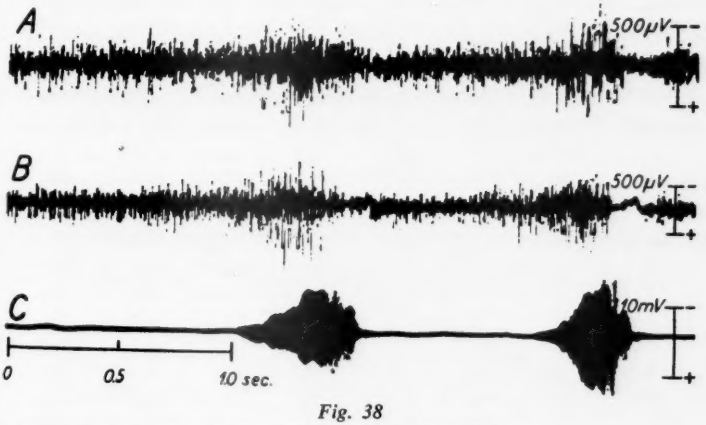

Fig. 38

Phonation of words with and without glottal catch ("stød").
A. Action potential pattern in the right cricothyroid muscle.
B. Action potential pattern in the right vocal muscle.
C. Microphone recording for "mord" (with "stød", to the left) and "mor" (without "stød", to the right). Frequency about 300 c.p.s. for both phonations.
Subject no. 13, a 65 year old man with normally movable vocal cords.

IV. CLOSURE OF THE LARYNGEAL APERTURE WITH COUGH, GLOTTAL CLICK AND SWALLOW

The amplitude of the action potential pattern was investigated during cough and during laryngeal movements similar to cough, i.e.

Table 11

The maximal amplitude of the action potential pattern, the microphone amplitude and the time interval between the onset of the change of the action potential pattern and the onset of tone as indicated by the microphone recording (△ t) in the cricothyroid, vocal and arytenoid muscles in subjects with normal larynx with phonation of words with and without glottal catch ("stød").

Experiment number	Activity at rest in μV	MOR (without glottal catch)			MORD (with glottal catch)		
		△t in msec.	Microphone amplitude in mV	Potential pattern amplitude in μV	△t in msec.	Microphone amplitude in mV	Potential pattern amplitude in μV
Cricothyroid muscle							
5	100	154	9,7	392	158	9,3	382
7	100	132	13,0	531	144	28,7	741
7	100	–	18,2	171	–	24,8	126
7	100	120	16,0	494	123	24,2	504
9	100	182	9,6	238	180	17,7	270
11	200	180	23,6	422	168	23,3	374
11	200	170	21,7	286	170	27,0	266
12	100	147	12,2	232	165	11,6	201
13	100	124	23,8	843	178	19,7	849
9	200	175	10,0	414	180	16,1	423
Vocal muscle							
5	150	165	9,7	450	168	9,3	516
7	80	–	13,0	218	140	28,5	364
7	80	–	18,2	258	205	24,8	242
7	80	140	16,0	318	123	24,2	360
9	60	197	9,6	300	190	17,7	442
11	200	181	23,6	384	205	23,3	426
11	200	178	21,7	262	140	27,0	284
12	100	150	12,2	507	160	11,6	375
13	250	188	23,3	1165	193	19,7	1360
Arytenoid muscle							
9	100	190	10	216	150	16,1	300
		Møller (without glottal catch)			Møller (with glottal catch)		
13	150	143	15	186	129	16,2	186

glottal click. Glottal click occurs when the glottis is closed and the air pressure in the trachea allowed to rise, so that the vocal cords are pressed apart. This is performed with open mouth without simul-

taneous movements of tongue or lips. The explosive opening of the glottis is not quite as vigorous as with cough. Both the cough and glottal click were recorded with the microphone simultaneously with the action potentials.

In addition the amplitude of the action potential pattern was investigated during swallow. The onset of swallow was signalled with a sound signal in the microphone and with a light signal on the action potential recording.

Figs. 39, 40, 41 and 42 give examples of the action potential pattern during cough, glottal click and swallow and Table 12 shows the amplitude of the action potential pattern. For comparison in every case the amplitude of the action potential pattern with phonation of "e" at a pitch of about 200 cycles per second is given.

In the cricothyroid, vocal and arytenoid muscles there was a considerable increase in the amplitude of the action potential pattern with cough, glottal click and swallow, and this increase occurred before the deflection on the microphone recording. The increase in the amplitude of the action potential pattern was greater with swallow than with phonation of "e"; there was no systematic difference

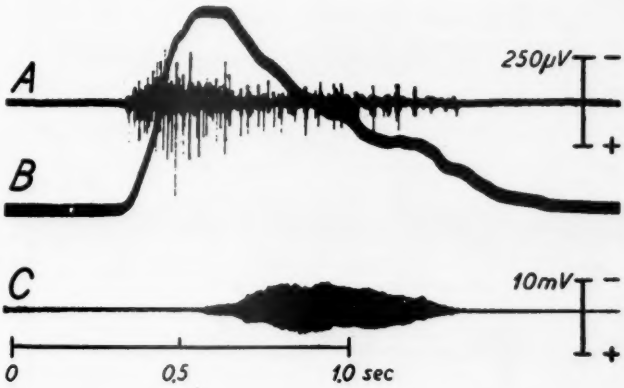

Fig. 39

Cough.
A. Action potential pattern in the left vocal muscle.
B. Mean action potential amplitude in the left vocal muscle.
C. Microphone recording.
Patient no. 56, a 64 year old woman with right vocal cord immovable in the paramedian position. The *left* vocal cord moved normally.

in the case of cough and glottal click. In many instances the amplitude of the action potential pattern during cough and glottal click decreased somewhat when the audible tone began as shown by the microphone recording, only to increase again and again to disappear about simultaneously with the disappearance of audible tone (Figs. 40 and 41).

The time interval between the onset of the increase in the action

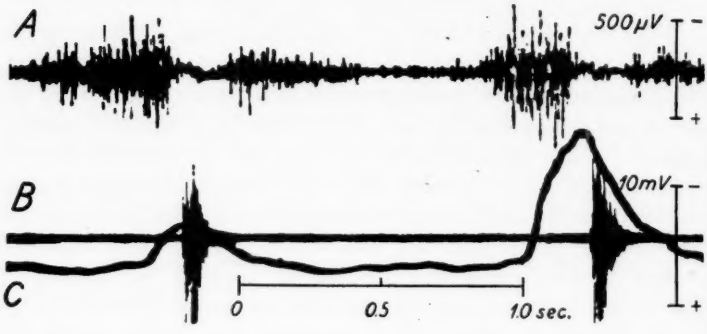

Fig. 40

Cough.
A. Action potential pattern in the left vocal muscle.
B. Microphone recording.
C. Mean action potential amplitude in the left vocal muscle.
Patient no. 60, a 42 year old man with the right vocal cord immovable in the intermediate position. The *left* vocal cord moved normally.

Fig. 41

Glottal click.
A. Action potential pattern in the left vocal muscle.
B. Microphone recording.
Subject no. 11, a 60 year old man with normally movable vocal cords.

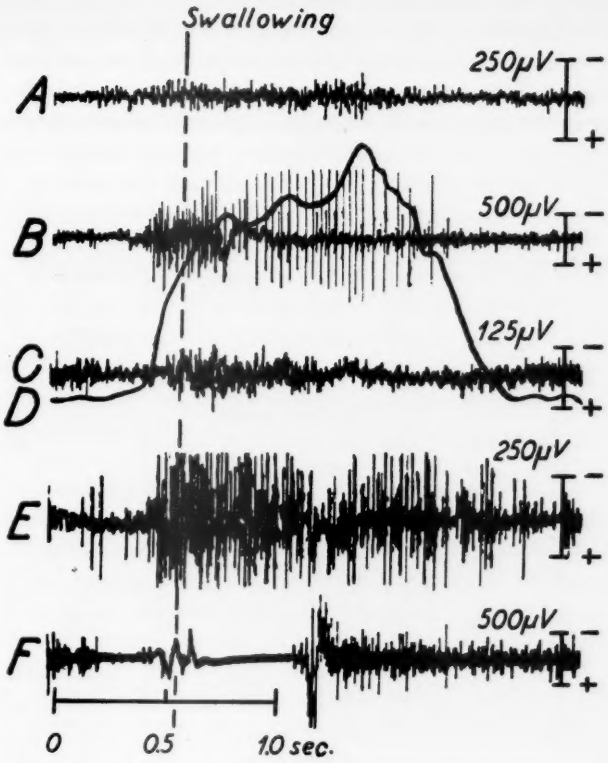

Fig. 42

Swallow.
The onset of swallow is indicated by the vertical stippled line.

A and E. Action potential pattern in the left cricothyroid muscle.
B. Action potential pattern in the left vocal muscle.
C. Action potential pattern in the left arytenoid muscle.
D. Mean action potential amplitude in the left vocal muscle.
F. Action potential pattern in the left posterior crico-arytenoid muscle.
A, B and D: patient no. 61, a 45 year old woman with the right vocal cord immovable in the paramedian position. The *left* vocal cord was normally movable.
C: subject no. 45, a 53 year old man with normally movable vocal cords.
E and F: patient no. 28, a 51 year old woman with the right vocal cord immovable in the paramedian position. The *left* vocal cord was normally movable.

Table 12

The maximal amplitude of the action potential pattern and the time interval between the onset of the change of the action potential pattern and the onset of tone as indicated by the microphone recording ($\triangle$ t) in the cricothyroid, vocal and arytenoid muscles in subjects with normal larynx during phonation of "e" (tone frequency about 200 cycles per sec.), cough, glottal click and swallow

Experiment number	Electrical activtiy at rest in μV	Phonation		Cough		Glottal click		Swallow
		$\triangle$ t in msec	Potential pattern amplitude in μV	$\triangle$ t in msec	Potential pattern amplitude in μV	$\triangle$ t in msec	Potential pattern amplitude in μV	Potential pattern amplitude in μV
				Cricothyroid muscle				
4	100	250	480					>480
5	120	310	172	1086	>580			353
7	200	253	534					530
8	150	252	366					>435
11	180	?	380			40	300	
16	150	100	225	730	>580			
17	140	?	277			366	300	>870
20	100	120	360	289	400	166	240	>550
21	120	250	330	124	1350			>553
26	100	300	540	260	240	198	300	
27	120	?	340	413	380	422	280	420
29	100	344	188			280	200	
				Vocal muscle				
4	100	300	300					664
5	200	343	542	1040	> 58			407
7	40	220	90					>508
11	200	?	450			280	>870	
45	15	180	23	440	90			>130
				Arytenoid muscle				
8	100		365	160	225	180	300	>435
11	100		220			46	260	
27	75	250	180	453	225	534	180	285
45	80		125	415	180	280	260	140

potential pattern's amplitude and the deflection on the microphone recording ($\triangle$ t) showed no systematic difference between phonation and glottal click. With cough the time interval was greater than with phonation and glottal click. Since it was not possible to signal accurately the time of onset of swallow the value of $\triangle$ t during swallow could not be determined.

In the posterior crico-arytenoid muscle there was a decrease of activity before cough, glottal click and swallow. In the case of cough and glottal click this decrease began approximately 0.35 seconds and ceased about 0.08 seconds before the onset of audible sound. Thus when the deflection appeared on the microphone recording normal electrical activity had been resumed in the posterior crico-arytenoid muscle.

V. DISCUSSION

Laryngeal text books give only scanty information as to function of the various intrinsic laryngeal muscles especially as to the significance of the individual muscles for the pitch, intensity and quality of tones produced. Contraction of the cricothyroid and vocal muscles is said to result in an increase in pitch; the arytenoid muscle is said not to contract during whispered speech; beyond this little information is available.

Weddell, Feinstein and *Pattle* (1944) investigated the intrinsic laryngeal muscles electromyographically in seven subjects. The muscles investigated were the thyro-arytenoid, the arytenoid and the lateral and posterior crico-arytenoid, both in normals and in patients with vocal cord paresis. The electromyographic findings were not however correlated with tone production. *Katsuki* (1950) investigated the cricothyroid muscle electromyographically in seven men, eight women and seven children and the vocal muscle in one patient who had undergone partial laryngectomy. Moreover *Husson* (1955) investigated the thyro-arytenoid and vocal muscles in two patients in the course of laryngeal operations.

In the present study an attempt was made to analyze the action potential pattern and the individual action potentials of the intrinsic laryngeal muscles to obtain information as to the function of these muscles in stretching and moving the vocal cords.

The motor unit potentials of the intrinsic laryngeal muscles were

usually di- or triphasic and of low amplitude (100–300 μV) and short duration (3–6 msec). This is in agreement with the findings of *Feinstein* (1946) (50–500 μV; 2–5 msec) and of *Husson* (1955) who found the individual action potentials to be of "low" amplitude and short duration (0.9–2 msec).

In all the adductor muscles included in the present investigation – the cricothyroid, vocal and arytenoid muscles – there was found a slight degree of "postural" electrical activity (up to 200 μV), while in the abductor muscle, the posterior crico-arytenoid muscle, a more pronounced "postural" electrical activity was present (200–500 μV). This is in agreement with *Weddell, Feinstein* and *Pattle* (1944) who report that the intrinsic laryngeal muscles have a "resting tone" attributable to "motor unit activity" since it is not possible to find "electrical silence" even when the subject holds his breath. Also *Husson* (1955) and *Fink, Basek* and *Epanchin* (1956) found electrical activity in the absence of phonation. *Katsuki* (1950) on the other hand, recording from the cricothyroid muscle, found no electrical activity in the absence of phonation in the majority of instances.

In view of the movements of the vocal cords with respiration (p. 18) it was to be expected that changes in electrical activity would occur during expiration. The investigations reported here have shown that during inspiration an increase in electrical activity occurs in all the adductor muscles investigated and a decrease in activity in the abductor muscle. With forced inspiration is there an increase in electrical activity in the abductor muscle in exceptional cases. The changes in electrical activity in the individual muscles do not begin simultaneously. The increase in electrical activity in the arytenoid muscle begins about 0.5 seconds before the increase in electrical activity in the cricothyroid muscle which precedes electrical activity in the vocal muscle by 0.9 seconds. In most instances all the intrinsic laryngeal muscles are involved in respiratory movements in contrast to the conception based on denervation experiments in animals (*Schech* 1873, *Rühlmann* 1874, *Bosworth* 1880, *Jelenffy* 1888, *Semon* 1890, *Grossmann* 1898). *Weddell, Feinstein* and *Pattle* (1944) reported similarly an increase in electrical activity during inspiration in many of the intrinsic laryngeal muscles. During expiration the electrical activity decreased, but did not disappear entirely. *Husson* (1955) found on the other hand an increase in electrical activity during expiration in the vocal muscle (about 40 mV) which decreased

(to about 15 mV) during inspiration *). *Fink, Basek* and *Epanchin* (1956), recording with an electroencephalograph, found during quiet respiration the same increase in electrical activity during inspiration as during expiration in the cricothyroid and the vocal muscles. During more violent respiration the increase in electrical activity was greater during expiration than during inspiration.

The function of contraction of the adductors of the larynx during inspiration is obscure. It may be that their contraction serves to steady the vocal cords and prevents the flapping which might otherwise result during inspiration. It is known that during inspiration the extrinsic laryngeal muscles contract, preventing descent of the larynx (*Mitchinson* and *Yoffey* 1947, *Andrew* 1955) with the trachea, which is pulled down two cm when the diaphragm contracts as shown by radiography (*Macklin* 1925). Incidental to the contraction of the extrinsic laryngeal muscles, the thyroid cartilage rocks backwards (*Schilling* 1937) and the vocal cords are thereby shortened, abducted and relaxed (*Michel* 1954), so that they could flap. That they do not, may be due to contraction of the adductor muscles during inspiration.

During phonation increased electrical activity was found in all the adductor muscles investigated, beginning 0.35–0.55 seconds before phonation. This is in agreement with *Katsuki* (1950) who found that electrical activity in the cricothyroid muscle begins before phonation.

When the increase in discharge frequency of a single motor unit during phonation could be determined, the resting frequency in the cricothyroid and vocal muscles was found to be up to about 12 per second and to increase before the onset of phonation to 15–30 per second, a frequency maintained during phonation. At the cessation of phonation the frequency fell rapidly to 12 per second or less. In occasional cases frequencies up to 50 per second were recorded during phonation but frequencies more rapid than this were never encountered. This is in agreement with the findings of *Katsuki* (1950) that frequencies between 3 and 40 per second are found in the cricothyroid muscle during phonation, depending on the pitch

*) Electrical activity of 15–40 mV measured with the needle electrodes applied by *Husson* have not previously been described in the literature even with maximal contraction, and appear on the basis of the present investigations hardly probable. A 10–20 times smaller amplitude seems more likely.

of the tone. *Husson* (1955) reported on the other hand a frequency in the vocal muscle of up to 500 per second. *Portmann, Robin, Laget* and *Husson* (1956) found a frequency of about 120 per second in the vocal muscle during phonation of *ut 2* (corresponding to "c", 129 cycles per second) and a frequency of about 200 per second during phonation of *la 2* (corresponding to "a", 217 cycles per second). The illustrated electromyographs on which these high frequencies were measured show an interference pattern of non-synchronized discharges from many motor units and do not in my opinion permit any conclusions as to frequency in the single units.

In normal vocal cords no significant increase in electrical activity occurs in the intrinsic laryngeal muscle with increasing intensity of phonation. This is in agreement with the findings of *Katsuki* (1950) that electrical activity in the cricothyroid muscle was only very slightly influenced when the intensity of tone increased. It is also in agreement with the view based on investigations of the excised larynx that tone intensity is primarily a function of the pressure of air in the trachea (*Johannes Müller* 1837, *Wyllie* 1866, *Jelenffy* 1888). A single patient with a laryngeal fistula after partial laryngectomy for cancer of the larynx allowing insertion of a needle electrode directly into the vocal muscle (*Katsuki* 1950) showed an increase in electrical activity with increasing intensity of tone, discernible in spite of much movement artifact. Under such abnormal conditions, however, this single finding can be given little weight. The patient could not control the pitch of tone but only its intensity. Were phonation begun with whispered voice and if tone first became audible with increasing intensity the result could be explained.

With rising pitch of phonation there was an increase in electrical activity in all the adductor muscles investigated, while electrical activity in the abductor muscles was inhibited. The increase in electrical activity was greater with increase of pitch within the same register than with a shift in register. Particularly in the vocal muscle there was little or no increase in electrical activity with shift of register. *Katsuki* (1950) found similarly an increase in electrical activity with increasing pitch in the cricothyroid muscle; with a change in register (a shift to falsetto in men) the electrical activity became irregular and this was attributed to an increase in the number of active motor units. A more likely explanation would seem to be the lesser increase in electrical activity with rise in pitch with a shift

in register than in the same register demonstrated in the investigations presented here. The action potential amplitude rose before phonation to nearly its value during phonation both in the cricothyroid and in the vocal muscle: the intrinsic laryngeal muscles assume the position and the degree of tension necessary for production of a tone of a certain pitch even before actual phonation.

Phonation of different vowels does not give any significant difference in electrical activity. The increase in electrical activity in the vocal muscle was relatively greater with phonation of words with than of words without glottal catch ("stød"), while in the cricothyroid muscle there was no difference. With different forms of tone initiation the change in electrical activity began earlier in the vocal muscle and was significantly greater with stopped than with even and breath attack. The difference in electrical activity was not significant in the cricothyroid muscle nor was the difference in activity significant between even and breath attack.

With whispered voice there was the increase in electrical activity found with ordinary phonation in all the adductor muscles investigated. The increase in activity was, however, somewhat less than with ordinary phonation in all muscles except the arytenoid where it was greater than with ordinary phonation, in disagreement with the assumption of *Pressman* (1942) that the arytenoid muscle does not contract with whispered voice. Similarly with "silent speech" there was an increase in activity in all the adductor muscles investigated but the increase was less than with ordinary phonation, being especially slight in the cricothyroid muscle. *Katsuki* (1950) found similarly an increase in activity in the cricothyroid muscle with "silent speech".

With cough, glottal click and swallow there appeared brief violent increases in activity in all the adductor muscles investigated and a corresponding inhibition in the abductor muscles. The increase in amplitude with swallow was usually greater than with phonation; there was no systematic difference with cough and glottal click. These movements are therefore produced by a significant exertion of muscle effort, and not as assumed by *Pressman* (1944) for cough and swallow, with a minimum of muscle effort.

How do the changes in electrical activity in the intrinsic laryngeal muscles found in this electromyographic study agree with conceptions as to the process by which tone is produced in the larynx?

The "neuro-muscular theory" (*Husson* 1955) postulates that the frequency of movement of the vocal cords with production of tone pitched up to 500 cycles per second is a result of a corresponding number of impulses from the brain transmitted via the nerves. Since in the present study the maximal frequency of a single motor unit during phonation was 50 cycles per second, in contrast to the 500 cycles per second reported by *Husson* (1955), this theory appears untenable. In agreement with the "aero-dynamic theory" is the finding in the present study that activity decreases slightly at the onset of phonation when suction is assumed to commence. The classical "myo-elastic theory" assumes that the frequency of vibration of the vocal cords with production of sound of a certain pitch is dependent primarily on the tension of the vocal cord. The results of the investigation presented here are in agreement with this assumption.

That the increase in activity is less with transition from a lower to a higher register than with increasing pitch in the same register is however not entirely explained or accounted for. An increase in the frequency of tone produced in the larynx can theoretically occur in any one of the following ways:

1) By means of increased tension of the vocal cords.
2) By shortening of the vocal cords.
3) By a decrease in the mass of the vocal cords.

It has been demonstrated that the vocal cords lengthen with increase in pitch (*Sonninen* 1954). This would of itself lower the frequency of the tone, but the increased passive tension compensates for this effect. Within the same register (for example chest register) the increase in pitch is accompanied by an increase in the active tension of the vocal cords (present investigation). As the pitch continues to increase, the point is finally reached at which the muscles can no longer increase the tension of the vocal cords. To produce a further increase in pitch the vocal cords must then either be shortened or their mass must be diminished. It has been claimed that the vocal cords are shortened with high tones by a "damping process", in as much as they lie in close apposition with each other over a small area posteriorly. With increasing pitch the area of apposition becomes larger and the anterior freely vibrating edge becomes smaller. This shortening of the vocal cords is said to explain the increase in pitch in the high registers (*Pressman* 1955). *Pressman* does not specify the frequency range of "high tones". That the vocal cords in the "upper

thin" register (p. 19) are shortened by a "damping process" appears demonstrated by the *Bell Telephone Laboratories,* High Speed Motion Film, (1940). The present investigation does not include investigations of phonation in the "upper thin" register. In the middle and head registers such a shortening of the vocal cords by a "damping process" is however not in agreement with the rod-shaped enlargement of the glottis in these registers observed by many investigators (*Lehfeldt* 1835, *Johannes Müller* 1837, *Rehti* 1896, *Musehold* 1898, *Trendelenburg* and *Wullstein* 1935, *Luchsinger* 1949). Confirmation is also lacking in the present investigation; inserting the electrode in different places in the vocal and thyro-arytenoid muscles during phonation in these registers there was not found such a high degree of electrical activity as would be expected if the vocal cords' free edges were shortened by a "damping process". It appears therefore unlikely that increase in pitch in middle and head registers occurs by means of a shortening of the vocal cords.

The mass of the vocal cords is composed of the vocal muscle and of the surrounding modified mucous membrane which contains abundant elastic tissue by which the mucous membrane is attached to the vocal muscle. Furthermore the sub-glottic portion of the larynx participates in the vibrations (*Smith* 1954), namely the conus elasticus in which certain of the fibres of the vocal and lateral crico-arytenoid muscles insert (*Mayet* 1955) and the mucous membrane loosely attached to and overlying the conus elasticus. Thus the mass of the vocal cords consists of a "coupled" system. With contraction of the vocal muscle the "stiffness" of the overlying mucous membrane increases and becomes greater than if the vocal muscle is relaxed. With chest register the vocal cords vibrate over their whole length, whereas with head register only the edges vibrate (*Luchsinger* 1949). There is thus hardly any doubt that the vibrating mass of the vocal cords is reduced with transition from a lower to a higher register, and this is also in agreement with the results of the present investigation. Whereas with increase in pitch within the same register electrical activity in the vocal muscle increased above that at rest, with transition from a lower to a higher register no increase in electrical activity occurred. This indicates that the vocal muscle does not show an additional contraction with transition to a higher register. Since the subject's voice was not changed by insertion of the 4 g needle electrode into the 318 mg vocal muscle (p. 41) the reduction in the

vibrating mass of the vocal cords with transition to a higher register does not depend upon whether the vocal muscle vibrates or not. Rather the "stiffness" of the mucous membranes must be increased, or the mass of the vibrating tissue decreased or both. With relaxation of the vocal muscle a lesser contraction of its pseudo-antagonist, the cricothyroid muscle, is required to increase tension of the free edge of the vocal cords. Therefore the increase in electrical activity in this muscle is less pronounced with transition to a higher register than with increase in pitch within the same register.

E. Patients With Vocal Cord Paresis.

I. THE POSITION OF THE PARETIC VOCAL CORD

Of 23 patients with unilateral paresis of the vocal cord the paretic cord was in the median or paramedian position in 17, in the intermediate position in 6. Four patients had bilateral paresis of the vocal cords with both vocal cords immovable in the median or paramedian position. Differentiation has not been attempted between the median and paramedian positions, since as emphasized by *Clerf* and *Suehs* (1941) it is often impossible to distinguish the two positions from each other particularly with unilateral paresis of the vocal cord. Median or paramedian positions are therefore referred to below as "paramedian position". Differentiation between paramedian and intermediate position offered no difficulty in the case discussed here.

The sex and age distribution of the patients and the etiology and duration of the paresis are shown in Table 13 and in the case histories on pages 128–138. The listing of patients in Table 13 is according to the position of the paretic vocal cord and according to the etiology of the paresis. With paretic vocal cords in the paramedian position a partial denervation caused by pressure or partial transection of the nerve in the lower portion of the neck or the upper portion of the mediastinum was the etiological factor; in three patients no etiology for the paresis could be determined. In patients with the paretic vocal cord in the intermediate position a partial denervation caused by pressure in the upper portion of the neck was the cause; in two patients no etiology could be demonstrated. It appears surprising that in 27 patients with vocal cord paresis there was not found total denervation in any, particularly since the vocal cord paresis in 10 patients followed thyroidectomy. The vocal cord paresis in the 10 patients cannot be due to complete interruption of

Table 13

Patients with vocal cord paresis

Number	Patients		Side with paresis	Position of paretic vocal cord	Paresis		
	Sex	Age in Years			Duration	Cause	
14	♀	38	right + left	Paramedian	7 months	Thyroidectomy	Partial denervation due to pressure or partial interruption of the nerve in the lower part of the neck or the upper part of the mediastinum.
39	♀	73	left	«	18 «	«	
40	♀	69	right	«	36 «	«	
42	♀	59	right + left	«	11 years	«	
47	♀	56	left	«	9 «	«	
52*	♀	43	(right) + left	«	24 months	«	
54	♀	70	left	«	11 years	«	
55	♀	53	right	«	9 «	«	
56	♀	64	right	«	1 month	«	
61	♀	45	right	«	54 months	«	
15*	♂	74	right + (left)	«	8 «	Goiter	
18	♀	41	left	«	19 years	«	
25	♂	70	left	«	42 months	Aorticaneurism	
35	♀	70	left	«	20 years	Glands in mediastinum	
48	♀	74	left	«	24 months	Cancer of the breast	
38	♂	54	right	«	1/2 month	Cancer of the esophagus	
58	♂	67	left	«	33 months	Cancer of the lung	
62	♂	30	left	«	1/2 «	Low neck trauma	
24	♀	57	left	«	>14 years	?	
28	♀	50	right	«	3 months	?	
33	♂	71	right	«	>8 years	?	
22	♀	58	right	Intermediate	17 months	Cancer of the base of the skull	Partial denervation due to pressure of the superior laryngeal nerve i. e. in the upper part of the neck.
36	♂	61	left	«	1 month	«	
49	♂	59	left	«	3 months	«	
60	♂	42	right	«	48 «	High neck trauma	
3	♂	20	left	«	1 1/2 «	?	
37	♀	62	left	«	12 «	?	

*) One vocal cord was immovable, the other could accomplish small rocking movements indicated by ( ).

the nerve but must have arisen either from partial interruption of the nerve or from pressure on the nerve (from ligature, hematoma or cicatricial changes).

With respect to the course of the vocal cord paresis this disappeared in one patient (no. 3) after having been present for $3^1/_2$ months. Six patients, all with cancer, died shortly after the investigation. The remaining 20 patients are all alive (1956) and their vocal cord paresis is unchanged.

II. THE DURATION OF THE SINGLE MOTOR UNIT POTENTIALS

Since the muscle action potentials show characteristic changes in neurogenic paresis the mean action potential duration of the intrinsic laryngeal muscles was compared on the paretic and on the normal side in as many of the patients as possible. Since only 5–10 different action potentials could be obtained from the same muscle, the patients were grouped according to the position of the paretic vocal cord and the mean action potential duration was calculated for the paretic and normal muscles in these groups (Table 14).

Table 14

The mean action potential duration in the intrinsic laryngeal muscles on the normal and the paretic side in patients with unilateral vocal cord paresis

Muscle	Position of the vocal cord	Action potentials on paretic side		Action potentials on normal side		Difference between duration on paretic and normal side in msec
		Mean duration in msec	Number	Mean duration in msec	Number	
Crico-thyroid	intermediate	5,9 ± 0,5	24	4,2 ± 0,7	11	1,7 ± 0,8
	paramedian	5,4 ± 0,4	41	5,1 ± 0,6	35	0,3 ± 0,6
Vocal	intermediate	5,3 ± 0,5	11	3,2 ± 0,3	19	2,1 ± 0,5
	paramedian	4,7 ± 0,3	88	3,5 ± 0,2	52	1,2 ± 0,3
Arytenoid [1])	paramedian	7,0 ± 0,8	14	4,7 ± 0,5	10	2,3 ± 0,9

[1]) The number of potentials in the posterior crico-arytenoid muscle and the arytenoid muscle in patients with immovable vocal cords in the intermediate position was so small that the values are not included in the table.

In the majority of instances the action potential duration was significantly longer in the paretic muscles, indicating a neurogenic paresis of these muscles. In patients with immovable vocal cord in

the paramedian position the cricothyroid muscle was an exception indicating the absence of a neurogenic paresis of this muscle in these patients.

III. THE ACTION POTENTIAL PATTERN IN THE ABSENCE OF PHONATION

The amplitude of the action potential pattern at rest was less in the intrinsic laryngeal muscles of patients with vocal cord paresis than in normal subjects, and on the paretic than on the normal side in patients with unilateral vocal cord paresis whatever the position of the paretic vocal cord.

With the greatest possible muscular relaxation and quiet respiration a slight degree of electrical activity with an amplitude of up to 100 μV was seen in the adductor muscles investigated in all patients with immovable vocal cords. Only the cricothyroid muscle in patients with immovable vocal cord in the paramedian position had an electrical activity as great as in normal muscles, up to 200 μV. In the posterior crico-arytenoid muscle electrical activity was more pronounced, with an amplitude of 100 to 300 μV in all patients with immovable vocal cords.

Respiration

Quiet Respiration

In patients with immovable vocal cords in the paramedian position the amplitude of the action potential pattern increased slightly during inspiration in the vocal and arytenoid and somewhat more in the cricothyroid muscles. The increase amounted to 20 to 200 μV in the vocal and arytenoid, to 20 to 500 μV in the cricothyroid muscles (Fig. 43). There was no change during expiration. Occasionally the amplitude of the action potential pattern did not change during respiration.

The increase in electrical activity began in the cricothyroid muscle about 0.9 seconds before the increase in electrical activity in the vocal muscle.

In patients with immovable vocal cords in the intermediate position there was no increase in electrical activity in the cricothyroid and vocal muscles during respiration and no inhibition of electrical activity in the posterior crico-arytenoid muscle.

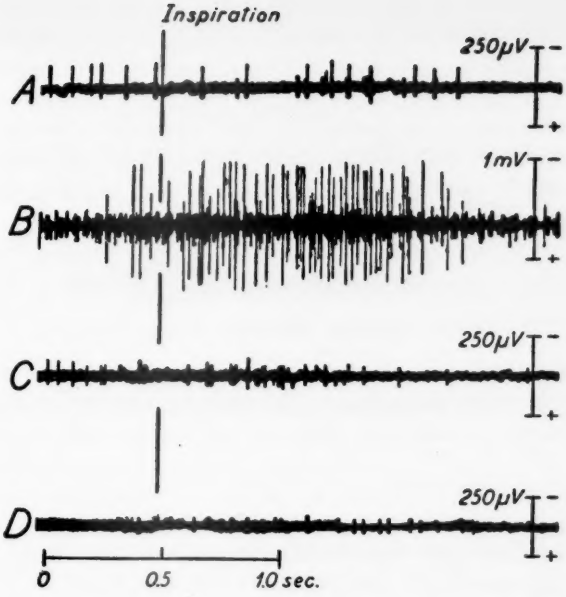

Fig. 43

Quiet respiration. The onset of inspiration is indicated by the vertical stippled line. A comparison between the normal (A and B) and paretic (C and D) sides.
A. Action potential pattern in the right cricothyroid muscle.
B. Action potential pattern in the right vocal muscle.
C. Action potential pattern in the left cricothyroid muscle.
D. Action potential pattern in the left vocal muscle.
Patient no. 25, a 71 year old man with the left vocal cord immovable in the paramedian position. The right vocal cord was normally movable.

Forced Respiration

In patients with immovable vocal cords in the paramedian position the amplitude of the action potential pattern increased by 200 to 300 μV in the vocal and arytenoid muscles during forced inspiration, by 200 to 600 μV in the cricothyroid muscle. Occasionally there was no change in the action potential pattern with forced inspiration. There was no change during forced expiration. No investigations were performed in patients with immovable vocal cords in the intermediate position during forced respiration.

In summarizing, the increase in electrical activity in the vocal and arytenoid muscles during inspiration was less in patients with paretic

vocal cord in the paramedian position than in normal subjects both in quiet and in forced respiration. In the cricothyroid muscle there was no significant difference in electrical activity with inspiration between normal subjects and patients with vocal cord paresis. In patients with immovable vocal cord in the intermediate position there was no increase in electrical activity during inspiration on the affected side in the cricothyroid and vocal muscles and no inhibition of electrical activity in the posterior crico-arytenoid muscle.

IV. PHONATION

a) **Electromyographic Findings During Phonation**

In all patients with immovable vocal cords the electrical activity increased during phonation in the adductor muscles investigated. Only in two patients was there no increase in electrical activity during phonation in the cricothyroid muscle. One patient (no. 3) had an immovable vocal cord in the intermediate position; the other (no. 54) in the paramedian position. The latter patient had a scar from thyroidectomy rather high in the neck which may have prevented placement of the needle electrode into the muscle.

In the abductor muscles investigated there was an inhibition of electrical activity during phonation in all patients with immovable vocal cord with the exception of a single patient (no. 56) in whom no activity could be demonstrated in the posterior crico-arytenoid muscle.

In patients with vocal cord paresis the action potential pattern during phonation often showed mixed activity or discharges of single motor units, whereas in normal subjects an interference pattern was usual (p. 54). The amplitude of the single action potentials could vary somewhat. This was due to small movements of the electrode during phonation whereby the distance to the active muscle fibres was changed and was not an expression of activity of new motor units or rotational activity (Figs. 44 and 45).

In patients with vocal cord paresis as in normal subjects the increase in electrical activity preceded the onset of audible sound by 0.35–0.55 seconds (Figs. 44 and 45).

In three patients with unilateral vocal cord paresis in the paramedian position there was a diminution of electrical activity during phonation in the cricothyroid muscle which usually showed an in-

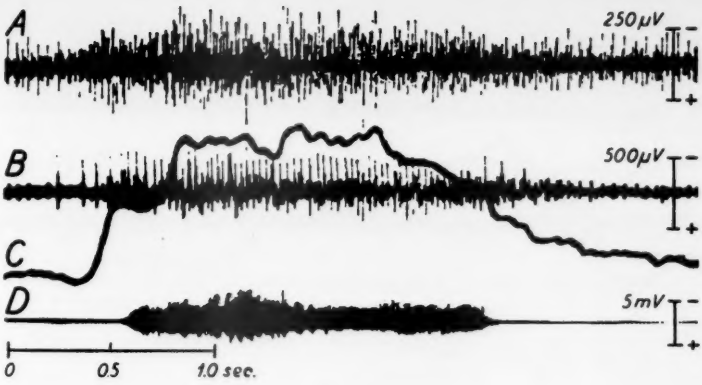

Fig. 44

Electrical activity in paretic muscles during phonation.
A. Action potential pattern in the left cricothyroid muscle.
B. Single motor unit discharges in the left vocal muscle.
C. Mean action potential amplitude in the left cricothyroid muscle.
D. Microphone recording.
Phonation: "e". Frequency 143 c.p.s.
Patient no. 58, a 67 year old man with the left vocal cord immovable in paramedian position. The right vocal cord was normally movable.

crease in electrical activity. This was seen both on the normal and on the paretic side; simultaneous recording from the *vocal* muscle on the contralateral side never demonstrated such a diminution of electrical activity during phonation.

This paradoxical diminution of electrical activity was only seen in simultaneous recording from the cricothyroid muscle on one side and the vocal muscle on the contralateral side. The three patients (nos. 35, 39 and 40) were all about 70 years old and were thus of an older average age than the patients as a whole (53 years); in no other respect did they differ from the other patients. Fig. 46 illustrates the inhibition of electrical activity in the cricothyroid muscle in two of these three patients.

Phonation with Open and Closed Tracheal Cannula

When normal subjects think of phonation an increase in electrical activity can be recorded electromyographically in the intrinsic laryngeal muscles, even though the air pressure in the trachea does

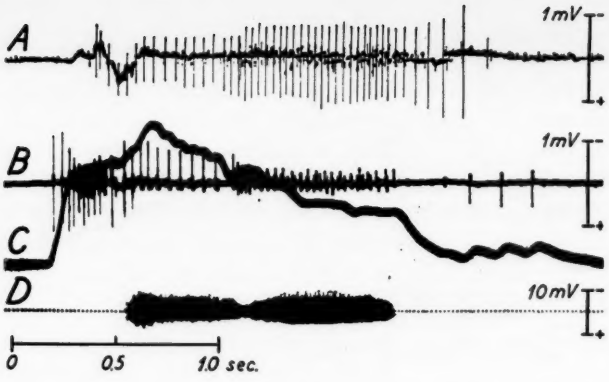

Fig. 45

Electrical activity in paretic muscles during phonation.
A. Single motor unit discharges in the left cricothyroid muscle.
B. Single motor unit discharges in the left vocal muscle.
C. Mean action potential amplitude in the left vocal muscle.
D. Microphone recording.
Phonation: "ahe". Frequency 222 c.p.s. for "a" and 285 c.p.s. for "e".
Patient no. 52, a 44 year old woman with both vocal cords immovable in the paramedian position. (The right vocal cord could however accomplish "rocking movements").

not rise as in ordinary phonation (cf. p. 72). It would have been of value to elucidate this point further by investigating tracheotomized subjects with normal freely movable vocal cords. Such subjects were unfortunately not at our disposal. However, two patients with permanent tracheal cannula were investigated; both had bilateral vocal cord paresis in the paramedian position. With an open tracheal cannula these patients had a poor, almost inaudible voice; when the tracheal cannula was closed the voice was good.

With phonation with a closed tracheal cannula the usual increase in electrical activity occurred before and during phonation in the cricothyroid and the vocal muscles. With an open tracheal cannula there was also an increase in electrical activity before and during phonation. The increase in electrical activity was however somewhat greater with a closed than with an open cannula, especially in the cricothyroid muscle, just as in the case of phonation with normal voice in comparison with whispered voice or "silent speech" (p. 72). Fig. 47 illustrates the action potential pattern of one of these patients.

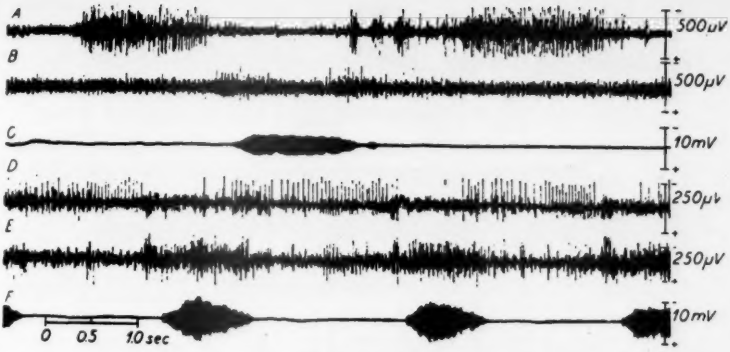

Fig. 46

Illustrations of inhibition of activity in the cricothyroid muscle during phonation in patients with unilateral vocal cord paresis. A–C: patient no. 35, D–F: patient no. 39.

A. Action potential pattern in the right cricothyroid muscle.
B. Action potential pattern in the left vocal muscle.
C. Microphone recording.
Phonation: "e". Frequency 333 c.p.s.
Patient no. 35, a 71 year old woman with left vocal cord immovable in paramedian position. The right vocal cord was normally movable.
D. Action potential pattern in the left cricothyroid muscle.
E. Action potential pattern in the right vocal muscle.
F. Microphone recording.
Phonation: "e". Frequency 182 c.p.s.
Patient no. 39, a 73 year old woman with the left vocal cord immovable in paramedian position. The right vocal cord was normally movable.

b) **The Discharge Frequency of the Single Motor Units**

In patients with neurogenic paresis of the extremity muscles the increase in discharge frequency of individual motor units with increasing effort can be followed since the normally functioning motor units in these cases are surrounded by fields of atrophic muscle fibres and there is therefore less chance of interference from neighbouring fibres (*Buchthal* and *Clemmesen* 1941). Similarly, the increase in frequency of a single motor unit during phonation has been followed in a number of patients with paresis of the vocal cord (Figs. 48 and 49).

In seven experiments the discharge frequency of single motor units in the cricothyroid muscle rose to at most 40 per second before the beginning of phonation and was maintained during phonation

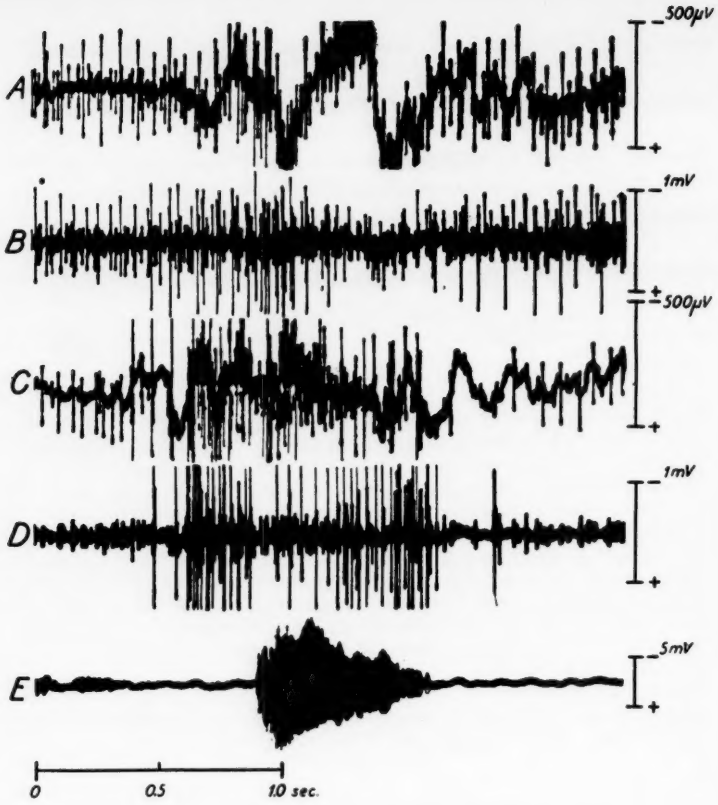

Fig. 47

Phonation with open (A, B) and closed (C, D) tracheal cannula.

A. Action potential pattern in the right cricothyroid muscle with open tracheal cannula.

B. Action potential pattern in the right vocal muscle with open tracheal cannula.

C. Action potential pattern in the right cricothyroid muscle with closed tracheal cannula.

D. Action potential pattern in the right vocal muscle with closed tracheal cannula.

E. Microphone recording.

Phonation: "e". Frequency 357 c.p.s.

Patient no. 42, a 59 year old woman with both vocal cords immovable in the paramedian position. The patient carried a permanent tracheal cannula.

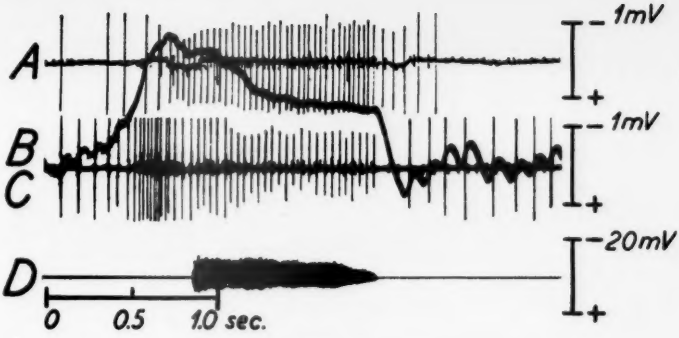

Fig. 48

Action potentials for a single motor unit during phonation.
A. Left cricothyroid muscle.
B. Left vocal muscle.
C. Mean action potential amplitude in the left vocal muscle.
D. Microphone recording.
Phonation: "e". Frequency 263 c.p.s.
Patient no. 52, a 44 year old woman with both vocal cords immovable in the paramedian position. (The right vocal cord could however accomplish "rocking movements"). The slight variation in the amplitude of the single potentials is due to movements of the electrode during phonation.

at 20–35 per second. Simultaneously with the end of phonation the frequency fell, and shortly after reached the resting value (Fig. 49). In ten experiments the frequency of single motor units in the vocal muscle rose from 5 to 50 per second before the onset of phonation. In a single experiment (Fig. 49) the frequency rose within a few milliseconds to 100 per second before the onset of phonation. During phonation the frequency was constant at 15–35 per second; simultaneously with the end of phonation it fell to about the resting value. It was not possible to follow single motor units in the arytenoid and posterior crico-arytenoid muscles.

Thus the discharge frequency for a single motor unit in the intrinsic laryngeal muscles in patients with vocal cord paresis was at most 40–50 per second; in a single instance the interval between two spikes corresponded to a discharge frequency of 100 per second.

c) Alteration in Tone Volume

The absence of a change in the amplitude of the action potential pattern with tone intensity on the normal side of the larynx in pa-

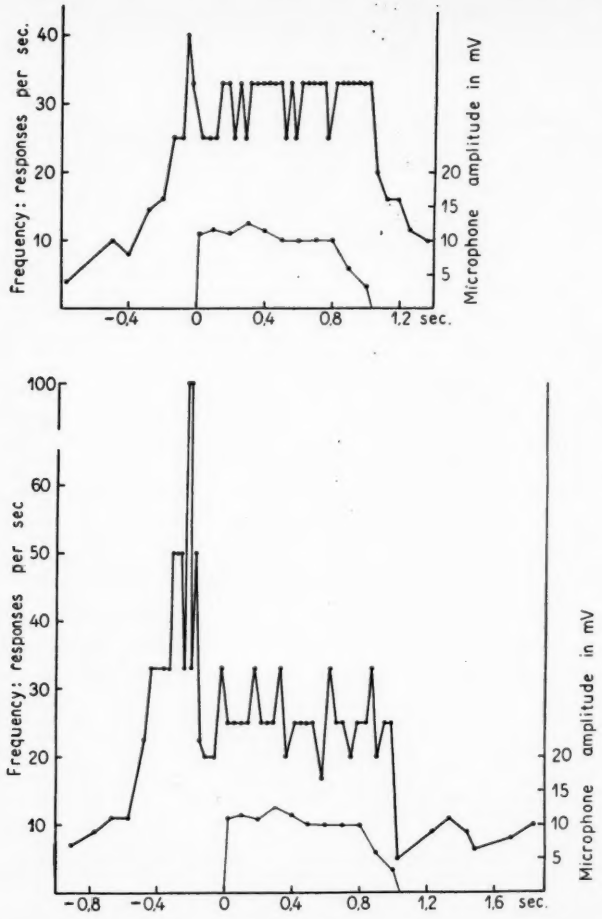

Fig. 49

Discharge frequency for a single motor unit shortly before, during and after phonation (thick line) and the amplitude of the microphone recording in mV (thin line) as a function of time in seconds for *the left cricothyroid muscle* (above) and *the left vocal muscle* (below) during the phonation illustrated in Fig. 48.
Phonation: "e". Frequency 263 c.p.c.
Patient no. 52, a 44 year old woman with both vocal cords immovable in the paramedian position. (The right vocal cord could however accomplish "rocking movements").

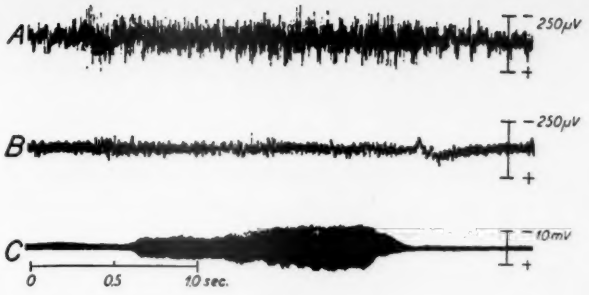

Fig. 50

A comparison between the action potential pattern during phonation with increasing tone volume in the normal (A) and the paretic (B) side of the larynx.
A. Action potential pattern in the left vocal muscle.
B. Action potential pattern in the right vocal muscle.
C. Microphone recording.
Phonation: "e". Frequency 250 c.p.s., constant during the whole phonation.
Patient no. 22, a 58 year old woman with the right vocal cord immovable in the intermediate position. The left vocal word was normally movable.

tients with unilateral vocal cord paresis has been described (p. 62). Similarly on the paretic side the amplitude of the action potential pattern did not increase significantly with the amplitude of the microphone recording. This was true in the cricothyroid, vocal and arytenoid muscles (Figs. 50 and 51).

d) Alteration in Tone Pitch

Changes with pitch in the amplitude of the action potential pattern on the normal side of the larynx in patients with unilateral vocal cord paresis have been described (p. 65).

In the cricothyroid (Fig. 52) and vocal (Figs. 53 and 54) muscles on the paretic side in patients with immovable vocal cords the maximal amplitude of the action potential pattern increased with pitch in all but one patient but the per cent increase was somewhat less than in normal subjects. The arytenoid muscle was investigated in two patients with bilaterally paretic vocal cords in the paramedian position. In one of these patients there was a slight increase and in the other a slight decrease in the amplitude of the action potential pattern with increasing pitch.

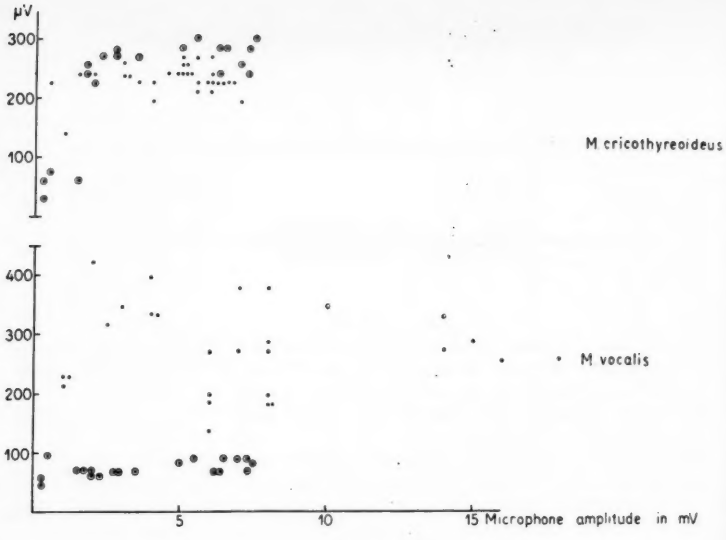

Fig. 51

The amplitude of the action potential pattern in the cricothyroid muscle (above) and the vocal muscle (below) in μV as a function of the amplitude of the microphone recording in mV for four different patients with paresis of the vocal cord during phonation of the vowel "e" with increasing tone volume.
• = patients with immovable vocal cord in the paramedian position.
⊙ = patient with immovable vocal cord in the intermediate position.
• (above) = patient no. 33. Frequency 334 c.p.s., a 71 year old man.
• (below) = patient no. 24. Frequency 303 c.p.s., a 58 year old woman.
⊙ = patient no. 22. Frequency 250 c.p.s., a 58 year old woman.

In summarizing, as in normal subjects and on the normal side of the larynx in patients with unilateral vocal cord paresis the amplitude of the action potential pattern was not a function of the intensity of tone in the affected muscles of patients with paretic vocal cord.

The amplitude of the action potential pattern did increase with increase in pitch on the paretic side in the laryngeal muscles but somewhat less than in normal subjects and on the normal side. The amplitude of the action potential pattern on the paretic side was usually less than half that of the normal side (Fig. 50). The cricothyroid muscle was an exception in patients with immovable vocal cords in the paramedian position. Here there was no difference in amplitude on the paretic and on the normal side.

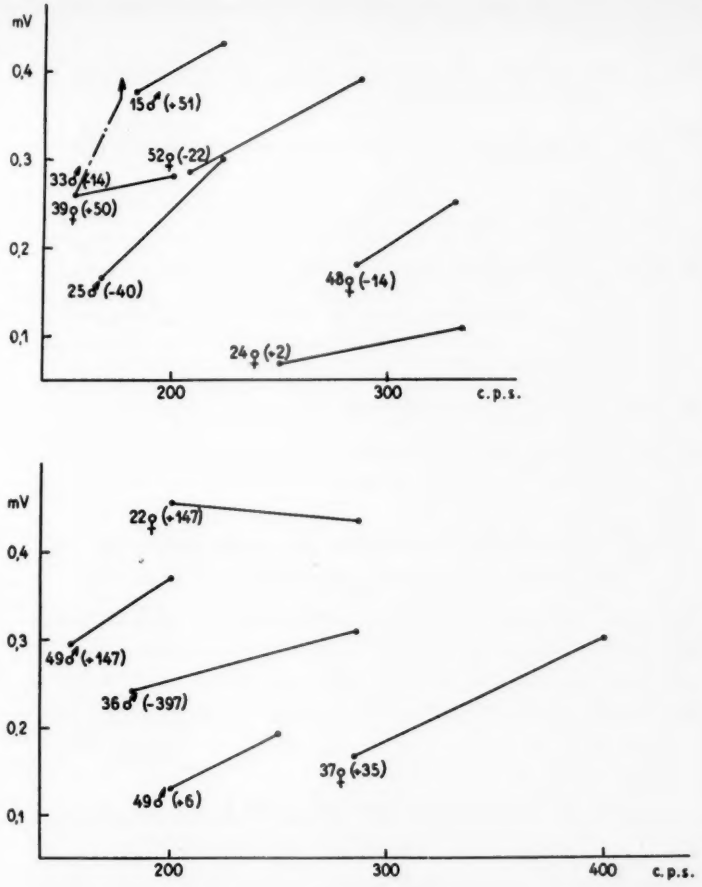

Fig. 52

Cricothyroid muscle.
The maximal amplitude in mV of the action potential pattern as a function of tone frequency in cycles per second for the cricothyroid muscle in patients with immovable vocal cords in the paramedian position (above) and intermediate position (below).
The numbers indicate the experiment number, the symbols the sex of the subjects and the numbers in parentheses the per cent change in tone volume from the low to the high tone. The stippled line indicate experiments in which the maximal amplitude for the high frequency could only be determined to be greater than the value indicated by the arrow.

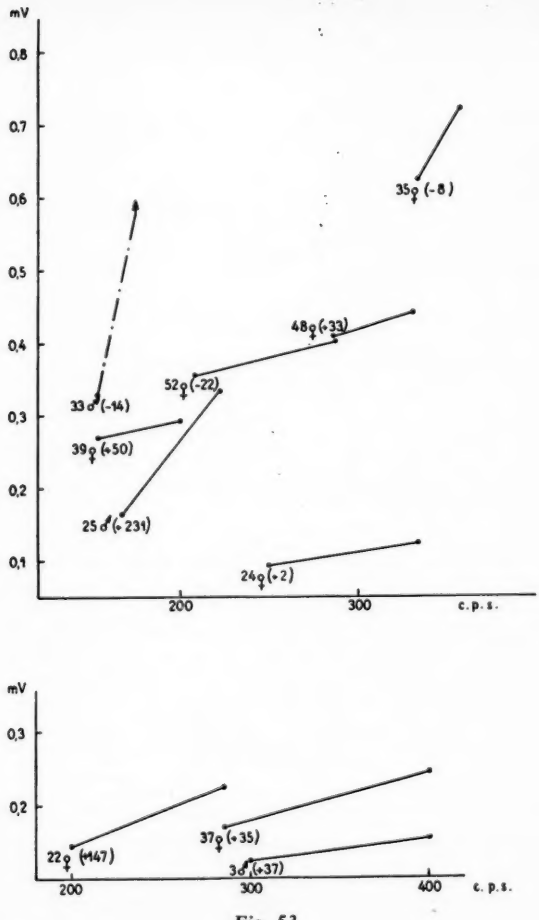

Fig. 53

Vocal muscle.
The maximal amplitude in mV of the action potential pattern as a function of tone frequency in cycles per second for the vocal muscle in patients with immovable vocal cord in the paramedian position (above) and intermediate position (below).
The numbers indicate the experiment number, the symbols the subject's sex and the numbers in parentheses the per cent change in tone volume from the low to the high tone. The stippled line indicate experiments in which it was only possible to determine the maximal amplitude for the high frequency as being greater than the value indicated by the arrow.

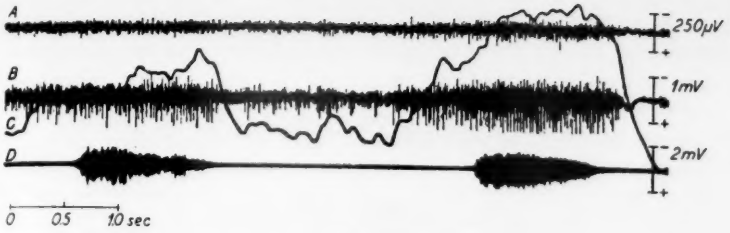

Fig. 54

The amplitude of the action potential pattern before and during phonation at different pitch in immediate succession.
A. Action potential pattern in the right cricothyroid muscle.
B. Action potential pattern in the right vocal muscle.
C. Mean action potential amplitude in the right vocal muscle.
D. Microphone recording.
Phonation: "e". Frequency 250 c.p.s. with the first phonation, 500 c.p.s. with the second phonation.
Patient no. 61, a 45 year old woman with the right vocal cord immovable in the paramedian position. The left vocal cord was normally movable.

e) The Amplitude of the Action Potential Pattern Before and During Phonation as a Function of Tone Pitch

As in normals and on the normal side in patients with unilateral vocal cord paresis the maximal and mean action potential amplitude rose before tone production (Fig. 54) to approximately its level during phonation on the paretic side in the cricothyroid, vocal and arytenoid muscles.

f) Comparison between the Electromyographic Findings on the Normal and Paretic Side of the Larynx

To investigate whether in patients with immovable vocal cords there is evidence of neurogenic paresis in the intrinsic laryngeal muscles, the action potential pattern was compared on the normal and the paretic side in patients with unilateral vocal cord paresis to obtain information as to the extent of loss of motor units. Furthermore the maximal amplitude was compared on the normal and the paretic side, since this is lower with paresis of neurogenic origin where fewer fibres are activated nearly simultaneously. Finally, denervation potentials were sought, appearing as spontaneous potentials of short duration and low amplitude.

In subjects with normally movable vocal cords there was found electrical activity in the intrinsic laryngeal muscles in the absence of phonation that is apparently at rest (p. 53). This "postural" electrical activity was a "motor unit" activity. The single potentials had a shorter duration and lower amplitude than action potentials recorded from the muscles of the extremities. Spontaneously appearing de-

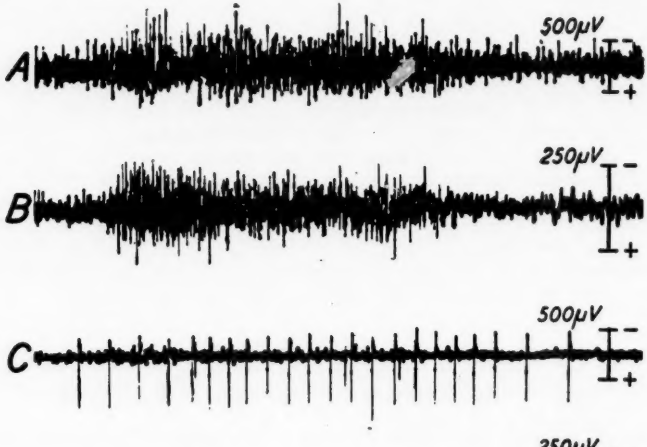

Fig. 55

A comparison of the action potential pattern during phonation in the normal (A, B) and paretic (C, D) sides of the larynx.
A. Action potential pattern in the left cricothyroid muscle.
B. Action potential pattern in the left vocal muscle.
C. Action potential pattern in the right cricothyroid muscle.
D. Action potential pattern in the right vocal muscle.
E. Microphone recording (the same for both sides).
Phonation: "e". Frequency 250 c.p.s.
Patient no. 22, a 58 year old woman with the right vocal cord immovable in the intermediate position. The left vocal cord was normally movable.

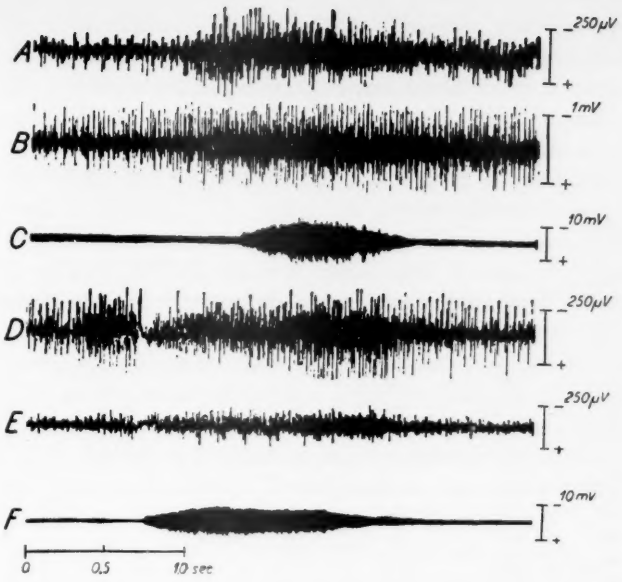

Fig. 56

A comparison between the action potential pattern during phonation in the normal (A, B) and paretic (D, E) sides of the larynx.
A. Action potential pattern in the right cricothyroid muscle.
B. Action potential pattern in the right vocal muscle.
C. Microphone recording.
D. Action potential pattern in the left cricothyroid muscle.
E. Action potential pattern in the left vocal muscle.
F. Microphone recording.
Phonation: "e". Frequency 200 c.p.s.
Patient no. 25, a 71 year old man with the left vocal cord immovable in the paramedian position. The right vocal cord was normally movable.

nervation potentials have similarly a short duration and low amplitude and can be difficult to differentiate from voluntary and involuntary "motor unit" activity in these muscles. However, denervation potentials have a still shorter duration than the motor unit potentials which appear in a normal laryngeal muscle. In the investigations reported here, denervation potentials were found in one muscle (vocal muscle, experiment no. 62). Although the vocal cord paresis in ten patients was a consequence of thyroidectomy, there was not any patient with a complete interruption of motor innervation to all intrinsic laryngeal muscles on one side.

Table 15

The action potential pattern on the normal and the paretic side of the larynx during phonation

N = normal side. P = paretic side. I = interfering activity.
M = mixed activity. S = single action potentials. – = no activity.

Experiment number	Phonation frequency in c. p. s.		Action potential pattern			
			Cricothyroid muscle		Vocal muscle	
	N	P	N	P	N	P
			Paramedian position			
18	?	?	I	I		
24	286	263	I	I	I	M
25	200	200	I	I	I	M
33	200	200	I	I	I	I
35	333	182	I	I	I	I
38	287	417	I	M	I	M
39	238	200	S	S	I	M
40	357	334	M	M	I	M
48	400	285	I	I	I	M
55	250	250	I	S	I	M
56	250	250	I	I	I	S
58	125	125	I	I	I	M
61	167	250	I	I	I	I
62	?	?	I	I	I	S
54	286	285			I	M
			Intermediate position			
3	230	300	I	–	I	S
22	250	238	I	S	I	M
36	222	208	I	S	I	S
37	284	286	I	M	I	M
49	200	200	I	M		
60	250	200	I	M	I	M

Of 23 patients with unilateral vocal cord paresis it was possible in most to compare the electromyographic findings in the normal and in the paretic cricothyroid and vocal muscles. For this comparison those experiments have been chosen where phonation of the vowel "e" was carried out, and where the pitch was approximately the same during both phonations. Such a comparison was not possible in the case of the other muscles.

The maximal amplitude of the action potential pattern was less

on the paretic than on the normal side (Fig. 55). The cricothyroid muscle was an exception in patients with immovable vocal cords in the paramedian position in that there was no difference in maximal amplitude on the two sides (Fig. 56). A comparison between the action potential pattern in the cricothyroid and the vocal muscles on the normal and the paretic side of the larynx during phonation is shown in Table 15. It appears that in the vocal muscle in the majority of instances there is evidence of loss of motor units in all patients with immovable vocal cord. In the cricothyroid muscle there was evidence of loss of motor units in all patients with immovable vocal cord in the intermediate position, but in the majority of instances there was no evidence of loss of motor units in patients with immovable vocal cord in the paramedian position.

In three patients the arytenoid and in two patients the posterior crico-arytenoid muscles were investigated on the normal side, and

Table 16

The number of intrinsic laryngeal muscles with and without evidence of neurogenic paresis as determined by a comparison of the action potential pattern on the normal and the paretic side of the larynx in patients with unilateral vocal cord paresis

The position of the paretic vocal cord	Muscle	The action potential pattern shows:		Total number of patients investigated
		evidence of neurogenic paresis	no evidence of neurogenic paresis	
Intermediate position:	Cricothyroid muscle	6	0	6
	Vocal muscle	5	0	5
	Arytenoid muscle	1	0	1
	Posterior crico-arytenoid muscle	1	0	1
Paramedian position:	Cricothyroid muscle	2	12	14
	Vocal muscle	11	3	14
	Arytenoid muscle	3	0	3
	Posterior crico-arytenoid muscle	2	0	2

all showed interference patterns. On the paretic side in other patients the arytenoid muscle was investigated in four, and the posterior crico-arytenoid in three, and all showed "mixed" activity or a degree of activity in which the single motor unit discharges could be discriminated.

Table 16 summarizes the findings with respect to the presence of signs of neurogenic paresis in muscles in the immovable half of the larynx.

In summary, a comparison of the appearance of the action potential pattern and of its amplitude on the normal and the paretic side in patients with unilateral vocal cord paresis indicates that all patients with paretic vocal cord in the intermediate position have evidence of neurogenic paresis in the intrinsic laryngeal muscles investigated. Most patients with a paretic vocal cord in the paramedian

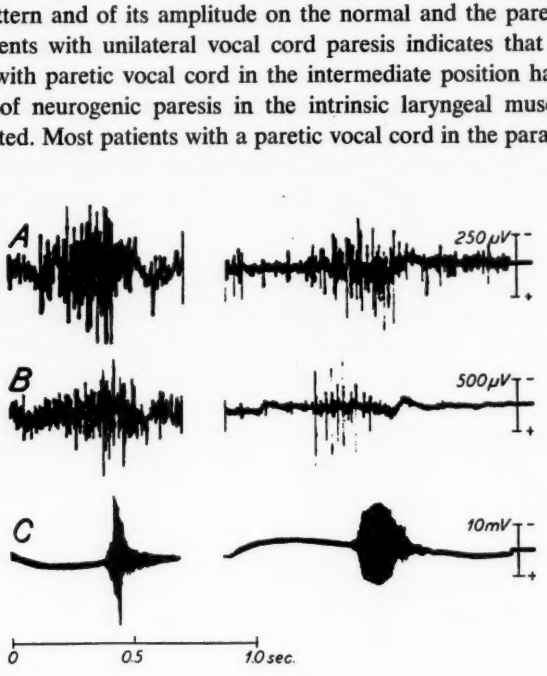

Fig. 57

Cough.
A comparison between the normal (to the left) and paretic (to the right) sides.
A. Action potential pattern in the cricothyroid muscle.
B. Action potential pattern in the vocal muscle.
C. Microphone recording.
To the left of the figure is the normal right half of the larynx, to the right of the figure is the paretic left half of the larynx.
Patient no. 25, a 71 year old man with the left vocal cord immovable in the paramedian position. The right vocal cord was normally movable.

position show evidence of neurogenic pareses in all the intrinsic laryngeal muscles with the exception of the cricothyroid muscle. This indicates that the cricothyroid muscle is not paretic or in any case less paretic in these patients.

V. CLOSURE OF THE LARYNGEAL APERTURE WITH COUGH AND SWALLOW

The amplitude of the action potential pattern during cough and swallow is given in Table 17, where the amplitude during phonation of vowels with a frequency of about 200 cycles per second is included for comparison. In the cricothyroid, vocal and arytenoid muscles there was a considerable increase in the amplitude of the action potential pattern with cough and swallow, and this increase occurred before the appearance of audible sound as indicated by the microphone recording. Compared with the increase in amplitude during phonation, the increase with cough and swallow was usually greater. This difference was, however, not statistically significant. Nor was there a significant difference between cough and swallow in the increase in amplitude. In many instances the amplitude of the action potential pattern decreased somewhat with the onset of audible tone in cough. Shortly after the amplitude rose again and returned to normal about simultaneously with the deflection on the microphone recording.

There was no difference between patients with immovable vocal cords in the paramedian and the intermediate positions in the change in amplitude of the action potential pattern with cough or swallow.

The time interval between the beginning of the change in electrical activity and the microphone recording ($\triangle$ t) was not different in phonation and in cough. Since it was not possible to indicate exactly the onset of swallow, $\triangle$ t could not be measured in the case of swallow.

The amplitude of the action potential pattern was lower on the paretic side than on the normal side in cough and swallow in patients with unilateral vocal cord paresis (Figs. 57 and 58). In the illustrated example from patients with immovable vocal cord in the paramedian position the electrical activity was especially diminished in the vocal muscle, not quite as much in the cricothyroid muscle.

In the posterior crico-arytenoid muscle there was an inhibition of

Table 17

The maximal amplitude of the action potential pattern and the time interval between the onset of the change of the action potential pattern and the onset of tone as indicated by the microphone recording ($\triangle$t) in patients with vocal cord paresis during phonation (tone frequency about 200 cycles per sec.), cough and swallow

Experiment number	Phonation		Cough		Swallow
	$\triangle$t in msec	Potential pattern amplitude in μV	$\triangle$t in msec	Potential pattern amplitude in μV	Potential pattern amplitude in μV
Cricothyroid muscle				Paramedian position	
14	470	370			113
24	367	67			100
25	250	165	275	400	
33	360	259	150	>480	415
39	260	260	526	>290	>290
48	397	180	376	200	225
56	200	90	286	>187	120
58	300	>435	222	>435	305
62	500	560			500
Cricothyroid muscle				Intermediate position	
22	260	456	360	390	375
60	200	70			47
Vocal muscle				Paramedian position	
24	427	95			590
25	280	165	250	1050	
33	345	330	770	>580	870
39	280	270	526	>435	345
48	373	408	428	260	320
58	400	450	255	>600	193
62	500	210			182
Vocal muscle				Intermediate position	
3	222	124			870
22	257	145	313	255	120
60	250	600			400
Arytenoid muscle				Paramedian position	
14	330	205			300
28	365	305	510	600	330

electrical activity before cough. This inhibition began and stopped before the onset of audible sound as indicated by the microphone recording. Thus at the time at which sound began as indicated by the microphone recording, electrical activity had returned to normal in the posterior crico-arytenoid muscle.

Patients with vocal cord paresis can seldom produce glottal click and its action potential pattern could not be investigated under these conditions.

VI. DISCUSSION

Transection of the recurrent nerve is thought to result in a paretic vocal cord in the paramedian position, whereas transection of both the recurrent and the superior laryngeal nerves results in a paretic vocal cord in the intermediate position (cf. p. 25). This difference in the position of the paretic vocal cords is assumed to be due in the main to the cricothyroid muscle, the only intrinsic laryngeal muscle innervated by the superior laryngeal nerve (*Burger* 1934, *Hofer* and *Jeschek* 1940, *Murtagh* 1945, *Negus* 1947, *Murtagh* and *Campbel* 1952). To determine the extent to which the positions in fact are determined by neurogenic paresis in different muscles the electrical activity of the intrinsic laryngeal muscles was compared on the normal and on the paretic side in patients with paretic vocal cord both in the paramedian and in the intermediate positions.

With the paretic vocal cord in the paramedian position there was no difference in the degree of electrical activity in the cricothyroid muscle between the paretic and the non-paretic side. The mean duration of the individual action potentials was the same on both sides and none of the electromyographic criteria showed evidence of neurogenic paresis in that muscle. With the paretic vocal cords in the intermediate position there was evidence of loss of motor units and prolonged action potential duration on the paretic side in contrast with the non-paretic side. In these cases, thus, there was evidence of neurogenic paresis supporting the assumption that the position of the paretic vocal cord depends on whether there is or is not paresis of the cricothyroid muscle.

In the other muscles (vocal, arytenoid and posterior crico-arytenoid muscles) in all patients with immovable vocal cord there was clear evidence of neurogenic paresis with loss of motor units

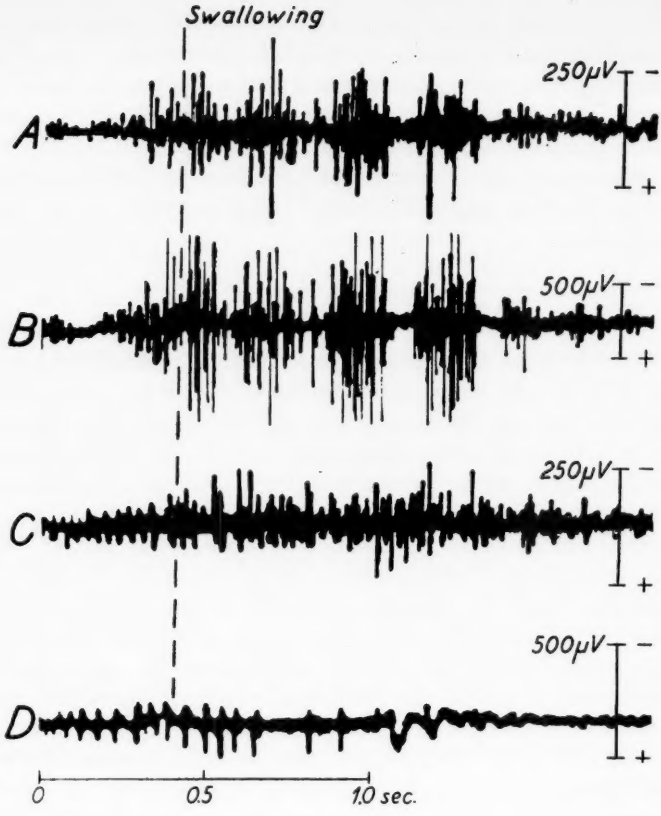

Fig. 58

Swallow.
A comparison between the normal (A, B) and paretic (C, D) sides.
The onset of swallow is indicated by the vertical stippled line.
A. Action potential pattern in the right cricothyroid muscle.
B. Action potential pattern in the right vocal muscle.
C. Action potential pattern in the left cricothyroid muscle.
D. Action potential pattern in the left vocal muscle.
Patient no. 58, a 67 year old man with the left vocal cord immovable in the paramedian position. The right vocal cord was normally movable.

and prolonged action potential duration. In addition the amplitude of the potential pattern during attempts at strong activity (high tones) was less than on the normal side.

Just as is the case in paresis of neurogenic origin in the skeletal musculature a marked loss of motor units in the paretic laryngeal muscles allows the frequency change of the action potential from single motor units to be followed with different degrees of activity. In the absence of phonation discharge frequencies of up to 11 per second were found. The frequency rose shortly before the onset of phonation, and was maintained during phonation at between 15 and 35 per second. At the cessation of phonation the frequency fell and rapidly reached the same values as before phonation. The highest discharge frequencies were about 50 per second with a tone frequency of 263 cycles per second; just once two potentials were separated by an interval corresponding to a frequency of 100 per second. As with normal subjects there was no correspondence between the frequency of the action potentials and the tone frequency as postulated by *Husson* (1950). In neurogenic paresis such a frequency measurement can be undertaken with considerable accuracy.

Weddell, Feinstein and *Pattle* (1944) investigated the action potentials of intrinsic laryngeal muscles in four patients. Two had "bilateral abductor paresis", presumably with the vocal cord immovable in the median position, due to thyroidectomy in one and to disseminated sclerosis in the other. In addition two patients were investigated with "unilateral complete vocal cord paresis", presumably with the vocal cord in the intermediate position due to a lesion of the neck in one and cause unknown in the other. The electromyographic findings are summarized in Table 18. *Weddell, Feinstein* and *Pattle* did not investigate the cricothyroid muscle. The results in two cases correspond to the results in the present investigation. In one patient there were denervation potentials in the posterior crico-arytenoid muscle on both sides, but normal electrical activity in the other muscle; such an elective denervation of single muscles was never found in the present study. The fourth patient had paresis of the vocal cords on the basis of anchylosis of the crico-arytenoid articulation as demonstrated by attempts at passive movements of the joints.

It has been claimed that motion is never regained in a vocal cord which has been paretic for more than six months even with recovery from the paresis since in the course of this time anchylosis has occurred in the crico-arytenoid articulation (*Delavan* 1903, *Hill* 1919, *Neumayer* 1921, *Jeschek* 1938). Others consider the ac-

Table 18

Weddell, Feinstein and Pattle's electromyographic investigations of patients with vocal cord paresis

Decr. act. = decreased activity.
Norm. act. = normal activity.
Fibr. pot. = fibrillation potentials (denervation potentials).

Muscle	The position of the paretic vocal cord			
	Bilateral paramedian position Case		Unilateral intermediate position Case	
	Number 1	Number 2	Number 3	Number 4
Right thyro-arytenoid	Decr. act.	Norm. act.		Norm. act.
Left » »	Decr. act.	Norm. act.		Norm. act.
Right lateral crico-arytenoid	Decr. act.		Fibr. pot.	Norm. act.
Left » »	Decr. act.		Norm. act.	Norm. act.
Arytenoid	Decr. act.	Norm. act.	Decr. act.	Norm. act.
Right posterior crico-arytenoid	Decr. act.	Fibr. pot.	Fibr. pot.	Norm. act.
Left » »	Decr. act.	Fibr. pot.		

companying changes which may occur of no importance for the immovability of the vocal cords (*Albrecht* 1929, *Krüger* 1929, *Hørbst* 1952, *Messerklinger* and *Probst* 1953). In all the patients discussed in the present study with paresis of the vocal cord the movability of the crico-arytenoid articulation was tested by passively moving the vocal process with a laryngeal rod and observing the movements by indirect laryngoscopy. It was found that the crico-arytenoid articulation could be moved with ease, so that in these instances it can be excluded that an anchylosis prevented movement of the vocal cords.

In patients with immovable vocal cords in the paramedian position in the majority of cases there occurred a slight increase in electrical activity in adductor muscles with respiration. This increase in electrical activity was less than in normal subjects, but occurred just as in normals during inspiration. In patients with immovable vocal cords in the intermediate position, on the other hand, no increase in electrical activity occurred in the adductor muscles during respi-

ration. This difference appears to be due to the different site of the lesion in the two forms of paresis. As mentioned above (p. 85) the trachea is drawn downwards during inspiration whereas the larynx is not or hardly not moved on account of the contraction of the other laryngeal muscles. Most of the extrinsic laryngeal muscles receive their innervation through the hypoglossal nerve. The present study has confirmed the importance of the superior laryngeal nerve for the position of the paretic vocal cords. A lesion of the recurrent nerve results in a paramedian position, while a lesion of both the recurrent and the superior laryngeal nerve results in an intermediate position. A lesion which simultaneously involves the recurrent and the superior laryngeal nerve must be situated in the upper portion of the neck where a simultaneous lesion of the hypoglossal nerve is likely. In all patients with immovable vocal cord in the intermediate position in whom electrical activity was shown to be absent during respiration there was neurological evidence of a lesion of the hypoglossal nerve. It must therefore be assumed that when no increase in electrical activity in the intrinsic laryngeal muscles can be demonstrated during respiration in patients with immovable vocal cords in the intermediate position this is due to a lesion of the hypoglossal nerve.

During phonation with increasing intensity there was no statistically significant increase in electrical activity in the adductor muscles investigated. During phonation with increasing pitch, on the other hand, electrical activity increased in the adductor muscles and was inhibited in the abductor muscles.

A comparison of the amplitude of the action potential pattern in the adductor muscles shortly before and during phonation with increasing pitch indicated that the amplitude rose before phonation to nearly its level during phonation. This indicates that the intrinsic laryngeal muscles of patients with vocal cord paresis are already set before phonation to the tension necessary to produce tone of a certain pitch just as is the case in subjects with a normal larynx.

With cough and swallow there was a brief increase in electrical activity in all the investigated adductor muscles.

A comparison between the action potential pattern amplitude on the normal and on the paretic side in patients with unilateral vocal cord paresis showed that the amplitude is less on the paretic than on the normal side.

In addition in the present investigation there was found no re-

lationship between the amplitude of the action potential pattern and the duration of the vocal cord paresis.

CONCLUSION

The present investigation has confirmed the assumption of the importance of the cricothyroid muscle for the position of paretic vocal cords. In patients with immovable vocal cords in the intermediate position there is evidence of neurogenic paresis in this muscle, while in patients with immovable vocal cords in the paramedian position there is no evidence of neurogenic paresis of it. In all the other intrinsic laryngeal muscles there is evidence of neurogenic paresis, both in patients with immovable vocal cords in the intermediate and in the paramedian positions.

In patients with a paretic vocal cord in the paramedian position, electrical activity increases in the adductor muscles during inspiration but is absent in patients with a paretic vocal cord in the intermediate position. The absence of such respiratory activity is probably attributable to a lesion of the hypoglossal nerve simultaneous with the recurrent and superior laryngeal nerve paresis responsible for the intermediate position of the paretic vocal cord.

Changes in electrical activity with and before phonation, with cough and with swallow resemble but are less than in normals or on the normal side.

Even long lasting vocal cord paresis was found not to be associated with anchylosis of the crico-arytenoid articulation.

SUMMARY

In the present study it was desired to determine in subjects with normally movable vocal cords the degree of electrical avtivity of the intrinsic laryngeal muscles as an indication of their function in producing movements of the vocal cords and determining their tension.

To contribute to a clarification of the neuro- and myogenic causes of vocal cord paresis the investigation was extended to include patients with vocal cord paresis.

The dimension of human intrinsic laryngeal muscles was determined on exised larynges. The number of muscle fibres in the different muscles was calculated, as well as the number of motor units

and the innervation ratio. In addition the elongation of the vocal cords was determined which results from the contraction of the cricothyroid muscle.

Non-paretic intrinsic laryngeal muscles were investigated electromyographically of 32 subjects with normally movable vocal cords and the normal side of 23 patients with unilateral vocal cord paresis. By means of concentric needle electrodes the action potentials were recorded from the cricothyroid, vocal, arytenoid, thyro-arytenoid and posterior crico-arytenoid muscles. Two muscles were investigated at one time and the subject's phonation was recorded by means of a microphone simultaneously with the action potentials. In addition the phonation was recorded on a tape recorder. The subject's voice was not essentially affected by placement of the electrodes in the intrinsic laryngeal muscles. In addition to recording the action potentials of the muscles directly, the mean action potential amplitude was recorded by means of an electronic device whose output current was proportional to the mean amplitude of the action potentials.

With phonation an increase in electrical activity was found in all the adductor muscles investigated and a decrease in electrical activity in the abductor muscles. The change in electrical activity began 0.35–0.55 seconds before audible sound as recorded by the microphone. When the discharge frequency of the single motor units could be measured, the maximum frequency was found to be 20–30 per second during phonation.

The single action potentials were di- or triphasic, their amplitude was 100–300 μV, and their duration 3–6 msec or about half the mean action potential duration in the brachial biceps muscle.

During phonation with increasing intensity there was no corresponding increase in electrical activity in the adductor muscles. With phonation with increasing pitch the increase in electrical activity was considerable as long as the increase in pitch occurred within the same register. If the increase in pitch occurred with a simultaneous shift in register the increase in electrical activity in most instances was only slight. The increase in electrical activity rose before phonation to nearly its level during the audible phonation as recorded by the microphone.

With phonation of different vowels there was no change in the action potential pattern.

With phonation with whispered voice and "silent speech" the increase in the amplitude of the action potential pattern in the adductor muscles was less than with ordinary voice.

During phonation with different forms of attack the amplitude of the action potential pattern in the vocal muscle was greater with stopped than with even or breath attack. The increase in electrical activity with stopped attack began relatively sooner than with the other forms of onsets.

The action potential amplitude in the vocal muscle was relatively greater with phonation of words with than without glottal catch ("stød").

During cough, glottal click and swallow there was a considerable increase in the amplitude of the action potential pattern in all the adductor muscles investigated, and this increase occurred before audible sound. The increase with cough began earlier than with phonation. In abductor muscles there was a reduction in electrical activity before cough, glottal click and swallow, and this reduction began about 0.35 seconds and ceased about 0.08 seconds before the microphone deflection.

In most subject's there was a slight degree of electrical activity at rest in the adductor muscles. Since this electrical avtivity did not disappear even when the subject held his breath, it must be considered a "postural" electrical activity. During inspiration this electrical activity increased but was unchanged during expiration. During forced respiration the inspiratory increase in electrical activity was more pronounced. In the abductor muscles there was a marked electrical activity at rest in all subjects. During inspiration this electrical activity decreased, but was unaffected during expiration.

The results of the investigations reported here do not support the *Husson* theory which assumes that the frequency of vibration of the vocal cords during phonation of a tone of a given pitch is a direct result of a corresponding number of impulses transmitted along the nerves, since the maximum discharge frequency in single motor units was only 30 per second, i.e. far below the vibrational frequency of the vocal cords.

The findings presented in this study are in agreement with the assumption that it is mainly the modified mucous membrane of the vocal cords which participates in the vibrations and that the vibrational frequency is determined by the active and passive tension

exerted by the different intrinsic laryngeal muscles as a result of differences in degree of their contraction.

Electromyography of the intrinsic laryngeal muscles on the paretic side was performed in 23 patients with unilateral vocal cord paresis, in 17 with the vocal cord in the paramedian and in 6 in the intermediate position. In addition, four patients with bilateral vocal cord paresis in the paramedian position were investigated.

The changes in the action potential pattern during respiration, before and during phonation of varying intensity and pitch, during cough and swallow were qualitatively the same as in subjects with normally movable vocal cords.

Comparing the action potential pattern, its amplitude and the duration af the single action potentials on the normal and paretic side indication was found of neurogenic paresis of all the intrinsic laryngeal muscles investigated on the paretic side. The only exception was the cricothyroid muscle in patients with immovable vocal cords in the paramedian position, where no evidence of neurogenic paresis was demonstrable.

The importance is emphasized of the cricothyroid muscle for the position of the paretic vocal cord in vocal cord paresis:

Lesion of n. recurrens without paresis of the cricothyroid muscle is associated with a paramedian position of the vocal cords; lesion of n. recurrens and of the superior laryngeal nerve results in a paresis also of the cricothyroid muscle and hence in an intermediate position of the vocal cords.

SAMMENDRAG

De hidtil anvendte metoder til undersøgelse af stemmelæbernes bevægelse og spænding under intonation tillader ikke med sikkerhed at skelne mellem, hvilke muskler der aktivt medvirker til at bevæge stemmelæberne, og hvilke muskler der passivt følger med, ligesom de ikke tillader at vurdere de enkelte musklers andel i stemmelæbernes spænding.

For at komplettere vor viden om de indre larynxmusklers betydning for stemmelæbernes bevægelse og spænding har man foretaget elektromyografiske undersøgelser af disse muskler hos forsøgspersoner med normalt bevægelige stemmelæber.

Ved elektromyografiske undersøgelser kan man få oplysninger om de indre larynxmusklers aktivitetsgrad. Herigennem kan man indirekte få oplysning om, hvilke muskler der medvirker ved stemmelæbernes bevægelser, og hvilke muskler der betinger stemmelæbernes spænding.

For at søge at bidrage til klarlæggelse af den neuro- og myogene årsag til stemmelæbepareser har man endvidere på tilsvarende måde undersøgt patienter med stemmelæbeparese.

Gennem måling på udtagne menneskelarynx har man bestemt de indre larynxmusklers størrelse samt stemmelæbernes forlængelse, når m. cricothyreoideus kontraheres, det vil sige, når afstanden mellem cartilago cricoideas forreste bue og cartilago thyreoideas underste kant mindskes. På grundlag af disse målinger og angivelser i litteraturen har man beregnet det totale antal muskelfibre i nogle af de indre larynxmuskler samt antallet af motoriske enheder og innervationsforholdet.

Der er foretaget *elektromyografiske undersøgelser af ikke-paretiske indre larynxmuskler* hos 32 forsøgspersoner med normalt bevægelige stemmelæber og hos 23 patienter med ensidig stemmelæbe-

parese. Hos sidstnævnte har man undersøgt musklerne på den normale side. Man har ved hjælp af nåleelektroder afledt aktionspotentialer fra m. cricothyreoideus, m. vocalis, m. arytenoideus, m. thyreoarytenoideus og m. crico-arytenoideus posterior, idet man har undersøgt 2 muskler af gangen og gennem en mikrofon registreret lyden samtidig med aktionspotentialerne. Endvidere har man optaget forsøgspersonernes intonationer på en Eltra-taperecorder. Forsøgspersonernes stemme påvirkes ikke væsentlig ved placering af nåleelektroder i de indre larynxmuskler. Foruden at registrere musklernes aktionspotentialer direkte har man registreret aktionspotentialernes middelværdi, der begge giver et udtryk for musklernes aktivitetsgrad, ved hjælp af en elektronisk anordning, som giver en udgangsspænding, der er proportional med middelamplituden af aktionspotentialerne.

I alle de undersøgte adductormuskler finder man en aktivitetsforøgelse og i abductormusklen en aktivitetsreduktion under intonation, som begynder 0,35–0,55 sek., før man gennem mikrofonen kan registrere hørbar lyd.

I de tilfælde, hvor det har været muligt at måle enkeltpotentialernes frekvens, viser det sig, at frekvensen for en enkelt motorisk enhed i de indre larynxmuskler i almindelighed ikke når over 20–30 pr. sek. under intonation.

De enkelte aktionspotentialer er di- eller trifasiske. Amplituden er 100–300 μV og varigheden 3–6 msek. Varigheden er således ca. 50 % af varigheden i m. biceps brachii.

Ved intonation med stigende tonestyrke optræder der ikke forøget aktivitet i adductormusklerne, men ved intonation med stigende tonehøjde kommer der en betydelig aktivitetstilvækst, dersom tonestigningen foregår indenfor samme register. Dersom tonestigningen foregår under samtidig registerskifte, er aktivitetstilvæksten derimod i de fleste tilfælde kun ringe. Ved undersøgelse af aktionspotentialmønstrets amplitude før og under intonation med forskellig tonehøjde ser man allerede før tonedannelsen en aktivitetsstigning, der svarer til den aktivitetsstigning, som ledsager tonedannelsen.

Ved intonation af forskellige vokaler ses der ingen forskelligheder i aktionspotentialmønstret.

Ved intonation med hviskestemme og "indre stemme" er aktionspotentialmønstrets amplitude i adductormusklerne mindre end ved almindelig stemme.

Ved intonation med forskellig ansats finder man, at aktionspotentialmønstrets amplitude i m. vocalis er større ved sprængansats end ved stand- og slagansats, og at aktivitetstilvæksten ved sprængansats kommer relativt tidligere end ved de andre former for ansats.

Ved intonation med og uden stød er aktionspotentialmønstrets amplitude i m. vocalis relativt større ved ord med stød end ved ord uden stød.

Ved hoste, adductionsknald og synkning finder man i alle de undersøgte adductormuskler en betydelig tilvækst i aktionspotentialmønstrets amplitude, og denne tilvækst kommer, før man kan iagttage noget mikrofonudslag. Tilvæksten ved hoste begynder tidligere end ved intonation. I abductormusklen ser man en aktivitetsreduktion før hoste, adductionsknald og synkning, og denne reduktion begynder ca. 0,35 sek. og ophører ca. 0,08 sek. før mikrofonudslaget.

Hos de fleste forsøgspersoner finder man i hvile en svag aktivitet i adductormusklerne. Da denne aktivitet ikke forsvinder, når forsøgspersonen holder vejret, må den opfattes som en »stillingsaktivitet«. Under inspirationen tiltager denne aktivitet, medens den er upåvirket af expirationen. Under forceret inspiration tiltager aktiviteten såvel mere som hyppigere end ved almindelig inspiration. I abductormusklen finder man i hvile en udtalt aktivitet hos alle forsøgspersonerne. Under inspiration reduceres denne aktivitet, medens den er upåvirket af expiration.

Resultaterne af de foreliggende undersøgelser bekræfter ikke den af *Husson* fremsatte teori, ifølge hvilken stemmelæbernes frekvens ved en bestemt tonehøjde er en direkte følge af et tilsvarende antal impulser modtaget gennem de innerverende nerver, idet man ikke finder en frekvens, der blot tilnærmelsesvis når 500 pr. sek. for en enkelt motorisk enhed.

Derimod er resultaterne af de foreliggende undersøgelser i overensstemmelse med antagelsen af, at det fortrinsvis er stemmelæbernes modificerede slimhinde, der deltager i svingningerne, og at svingningsfrekvensen bestemmes af den aktive og passive spænding, som de forskellige indre larynxmuskler ved deres varierende kontraktion giver slimhinden.

Der er foretaget *elektromyografiske undersøgelser af paretiske indre larynxmuskler* hos 23 patienter med ensidig stemmelæbeparese (17 i paramedianstilling og 6 i intermediærstilling) og hos 4 patienter med dobbeltsidig stemmelæbeparese i paramedianstilling.

Ved undersøgelse af aktionspotentialmønstret i hvile, under respiration, før og under intonation med variation af tonestyrke og tonehøjde samt under hoste og synkning, finder man hos patienter med ubevægelige stemmelæber i paramedian- og intermediærstilling ganske tilsvarende forandringer som hos forsøgspersoner med normal bevægelige stemmelæber.

Ved sammenligning mellem aktionspotentialmønstret, aktionspotentialmønstrets amplitude og de enkelte aktionspotentialers varighed i den normale og i den paretiske side hos patienter med ensidig stemmelæbeparese finder man tegn til neurogen parese i alle de undersøgte indre larynxmuskler på den paretiske side både hos patienter med ubevægelige stemmelæber i paramedian- og intermediærstilling. En undtagelse herfra danner m. cricothyreoideus hos patienter med ubevægelige stemmelæber i paramedianstilling, hvor der ikke kan påvises tegn til neurogen parese. En tilsvarende sammenligning mellem patienter med stemmelæbeparese og normale forsøgspersoner giver samme resultat.

Den foreliggende undersøgelse bekræfter således formodningen om m. cricothyreoideus betydning for de paretiske stemmelæbers stilling ved stemmelæbepareser.

Case Histories

The sensitivity of the larynx of each patient was tested by touching the mucous membrane at different points with a cotton-tipped laryngeal swab and obse. ving the reflex movements in the larynx by indirect laryngoscopy. In most patients it was found that the sensitivity was the same on the paretic and on the normal side, and this is not indicated for each patient. The few cases are noted where the sensitivity was different on the two sides.

The mobility of the crico-arytenoid articulation was investigated on the paretic side by moving the vocal process with a laryngeal swab after anesthetizing the mucous membrane with a 0.5 per cent solution of tetracain with added adrenalin. None of the patients had evidence of anchylosis of the crico-arytenoid articulation.

Whenever, in the brief case histories summarized below, mention is made of an "immovable" vocal cord, a vocal cord is meant which cannot be seen by indirect laryngoscopy to move from its position during respiration, phonation or cough.

By complete blood investigation is meant hemoglobin per cent, red and white cell count, differential count and color index.

By action potential amplitude is meant the maximum amplitude of the action potential pattern at phonation with high pitch.

The succession of case histories is as in Table 13 on page 92.

Patient no. 14

E. J. Female. 38 years.

Cause of vocal cord paresis: Thyroidectomy.

Laryngoscopy on April 10, 1953, showed no abnormalities, in particular both vocal cords were freely movable. On April 16, 1953, a subtotal thyroidectomy was performed. During operation there developed increasing respiratory stridor so that an inferior tracheotomy had to be performed. Laryngoscopy on April 17, 1953, revealed immovable vocal cords in the paramedian position on both sides. The glottis was 2–3 mm in width.

On January 30, 1954, electromyography of the laryngeal musculature showed evidence of loss of motor units in the left arytenoid muscle but not in the left cricothyroid muscle.

On laryngoscopy both vocal cords were found to be immovable in the paramedian position. The glottis was 2–3 mm in width. The vocal cords were not excavated. The patient still had a tracheal cannula. The voice was good (not hoarse) when the patient closed off the cannula.

Patient no. 39

L. R. Female. 73 years.

Cause of vocal cord paresis: Thyroidectomy.

In 1935 a subtotal thyroidectomy was performed. There was no hoarseness after this operation. On October 25, 1952, re-operation was performed. After this operation the voice became hoarse and laryngoscopy revealed the left vocal cord to be immovable in the paramedian position.

On May 15, 1954, electromyography of the laryngeal musculature showed evidence of loss of motor units in the left vocal but not in the right vocal or right or left cricothyroid muscles. The action potential amplitude in the left vocal muscle was about half that in the right vocal muscle. The amplitude in the right and left cricothyroid muscles was the same.

On laryngoscopy the left vocal cord was found to be immovable in the paramedian position but not shortened or excavated. The right vocal cord was normally movable. The voice was deep but not hoarse.

Patient no. 40

H. J. Female. 69 years.

Cause of vocal cord paresis: Thyroidectomy.

On July 13, 1951, a subtotal thyroidectomy was performed. Hoarseness developed after operation. Laryngoscopy revealed the right vocal cord to be immovable in the paramedian position.

On May 15, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the right vocal but not in the left vocal or right or left cricothyroid muscles. The action potential amplitude in the right vocal muscle was about one third that in the left vocal muscle. The action potential amplitude in the right and left cricothyroid muscles was the same.

The right vocal cord was immovable in the paramedian position, not shortened or excavated. The left vocal cord was normally movable. The voice was slightly hoarse.

Patient no. 42

M. M. Female. Age 59 years.

Cause of vocal cord paresis: Thyroidectomy.

In 1943 a subtotal thyroidectomy was performed. After operation severe respiratory stridor developed and tracheotomy was required. On laryngoscopy both vocal cords were found immovable in the paramedian position.

On May 22, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the right and left vocal muscles but not in the right cricothyroid muscle.

Both vocal cords were immovable in the median position but not excavated; the glottis was entirely closed. The patient still had a tracheal cannula. The voice was good and not hoarse when the cannula was occluded.

Patient no. 47

M. R. Female. Age 56 years.

Cause of vocal cord paresis: Thyroidectomy.

In 1943 a subtotal thyroidectomy was performed. There was no hoarseness after this operation. In 1954 re-operation was performed. Thereafter the voice became hoarse, and laryngoscopy demonstrated an immovable vocal cord in the paramedian position on the left.

On July 22, 1954, electromyography of the laryngeal muscles showed no evidence of loss of motor units in the left cricothyroid and right vocal muscles.

On laryngoscopy the left vocal cord was found immovable in the paramedian position, not shortened or excavated. The right vocal cord was normally movable. The voice was slightly hoarse.

Patient no. 52

A. J. Female. Age 43 years.

Cause of vocal cord paresis: Thyroidectomy.

On August 4, 1952, subtotal thyroidectomy was performed. Hoarseness developed after operation. On laryngoscopy both vocal cords were seen immovable in the paramedian position.

On July 31, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the right and left vocal and the right arytenoid muscles but not in the right cricothyroid muscle. The left cricothyroid muscle showed a variable potential pattern but usually without evidence of loss of motor units.

On laryngoscopy both vocal cords were found immovable in the paramedian position, although the right could perform small rocking movements about the paramedian position. There was no excavation of the vocal cords. The glottis was 2–4 mm in width. The voice was slightly hoarse.

Patient no. 54

L. L. Female. Age 70 years.

Cause of vocal cord paresis: Thyroidectomy.

On March 2, 1943, subtotal thyroidectomy was performed. Hoarseness developed after operation. On laryngoscopy the left vocal cord was found immovable in the intermediate position.

On August 5, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the left vocal muscle but not in the right vocal or left cricothyroid muscles. The findings in the right cricothyroid muscle were somewhat variable but usually without evidence of loss of motor units. The action potential amplitude in the left vocal muscle was about half that in the right vocal muscle.

Laryngoscopy demonstrated the left vocal cord immovable in the paramedian position, not shortened or excavated. The right vocal cord was normally movable. The voice was not hoarse.

Patient no. 55

A. T. Female. Aage 53 years.

Cause of vocal cord paresis: Thyroidectomy.

On July 1, 1945, subtotal thyroidectomy was performed. After operation hoarseness developed. Laryngoscopy demonstrated the right vocal cord to be immovable in the paramedian position.

On August 5, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the right vocal and right cricothyroid muscles but not in the left vocal and left cricothyroid muscles.

On laryngoscopy the right vocal cord was found immovable in the paramedian position, not shortened or excavated. The left vocal cord was normally movable. The voice was not hoarse.

Patient no. 56

A. H. Female. Age 64 years.

Cause of vocal cord paresis: Thyroidectomy.

On July 10, 1954, subtotal thyroidectomy was performed. Hoarseness developed after operation. On laryngoscopy the right vocal cord was immovable in the paramedian position.

Electromyography of the laryngeal muscles on August 7, 1954, showed evidence of loss of motor units in the right vocal and posterior crico-arytenoid muscles but not in the left vocal, posterior crico-arytenoid nor in the right and left cricothyroid muscles. The action potential amplitude in the right vocal muscle was about one twentieth that in the left vocal muscle. The amplitude in the right and left cricothyroid muscles was the same.

On laryngoscopy the right vocal cord was found immovable in the paramedian position, not excavated or shortened. The left vocal cord was freely movable. The voice was slightly hoarse.

Patient no. 61

A. H. Female. Age 45 years.

Cause of vocal cord paresis: Thyroidectomy.

In February, 1950, subtotal thyroidectomy was performed. Hoarseness developed after operation. Laryngoscopy demonstrated the right vocal cord to be immovable in the paramedian position.

On August 21, 1954, electromyography of the laryngeal muscles showed no evidence of loss of motor units in the right and left vocal or in the right and left cricothyroid muscles.

On laryngoscopy the right vocal cord was found immovable in the paramedian position, slightly shortened and excavated. The left vocal cord was freely movable. The voice was deep and somewhat hoarse.

Patient no. 15

P. V. Male. Age 74 years.

Cause of vocal cord paresis: Goiter.

On May 17, 1953, the patient suddenly became hoarse. On laryngoscopy both vocal cords were found in the paramedian position, the right immovable, while the left could perform small rocking movements about the paramedian position.

X-ray examination of the mediastinum showed a round plum-sized shadow, invading the right lung field. Roentgen diagnosis: Intrathoracic tumor.

Neurological investigation demonstrated no certain abnormalities.

Wassermann reaction: negative.

Complete blood examination: normal.

Basic metabolic rate: normal.

The thyroid gland was normal to palpation.

On January 30, 1954, electromyography of the laryngeal muscles showed

evidence of loss of motor units in the right and left vocal and in the left arytenoid muscles but not in the right and left cricothyroid muscles.

On laryngoscopy both vocal cords were found in the paramedian position, the right immovable, while the left could perform small rocking movements about the paramedian position. The vocal cords were not shortened or excavated. The voice was slightly hoarse.

Patient no. 18

H. E. Female. Age 41 years.

Cause of vocal cord paresis: Goiter.

Had never noticed hoarseness. In 1935 during pregnancy an enlargement of the thyroid gland developed on the left and disappeared after roentgen treatment. On January 29, 1954, laryngoscopy demonstrated an immovable left vocal cord in the paramedian position.

X-ray of the mediastinum showed no abnormalities.

Neurological investigation: no abnormality.

Wassermann reaction: negative.

Complete blood investigation: no abnormalities.

The thyroid gland was normal to palpation.

On February 13, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the left posterior crico-arytenoid muscle but not in the right arytenoid or right or left cricothyroid muscles.

On laryngoscopy the left vocal cord was found immovable in the paramedian position, not shortened or excavated. The right vocal cord was normally movable. The voice was not hoarse.

Patient no. 25

C. J. Male. Age 70 years.

Cause of vocal cord paresis: Aortic aneurysm.

On October 11, 1950, the patient suddenly became hoarse. On laryngoscopy the left vocal cord was found immovable in the midline.

X-ray examination of the mediastinum demonstrated a pronounced enlargement of the heart and especially of the aorta.

Neurological investigation: no abnormality.

Wassermann reaction: negative.

Complete blood investigation: no abnormality.

No abnormalities found on palpation of the neck.

On March 6, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the left vocal muscle but not in the right vocal or right or left cricothyroid muscles. The action potential amplitude in the left vocal muscle was about one fifth that in the right vocal muscle. The amplitude in the right and left cricothyroid muscles was the same.

On laryngoscopy the left vocal cord was found immovable in the midline, not excavated or shortened. The right vocal cord was normally movable. The voice was slightly hoarse.

Patient no. 35

K. A. Female. Age 70 years.

Cause of vocal cord paresis: Enlarged glands in the mediastinum.

In 1934 the patient suddenly became hoarse. Laryngoscopy revealed the left vocal cord to be immovable in the paramedian position.

X-ray examination of the mediastinum demonstrated a large calcified gland under the arch of the aorta.

Neurological investigation: no abnormality.

Wassermann reaction: negative.

Complete blood investigation: no abnormality.

No abnormality on palpation of the neck.

On April 10, 1954, electromyography of the laryngeal muscles showed no evidence of loss of motor units in the right vocal or in the right and left cricothyroid muscles. The left vocal muscle showed somewhat variable findings but usually without evidence of loss of motor units.

On laryngoscopy the left vocal cord was found immovable in the paramedian position, slightly shortened but not excavated. The right vocal cord was freely movable. The voice was slightly hoarse.

Patient no. 48

A. M. Female. Age 74 years.

Cause of vocal cord paresis: Metastases to the mediastinal glands from cancer of the breast.

In 1950 an amputation of the left breast was performed on account of cancer. On September 1, 1952, the patient suddenly became hoarse. On laryngoscopy the left vocal cord was found immovable in the paramedian position.

X-ray examination of the lungs and mediastinum demonstrated spotty infiltrations in the right lung base, presumably metastases. The trachea was displaced to the right by metastases to the left paratracheal glands.

Neurological investigation: no abnormality.

Wassermann reaction: negative.

Complete blood examination: no abnormality.

On physical examination glandular metastases in the left axilla and above the left clavicula were found.

On July 24, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the left vocal but not in the right vocal or right or left cricothyroid muscles. The action potential amplitude in the left vocal muscle was about half that in the right vocal muscle. The amplitude in the right and left cricothyroid muscles was the same.

On laryngoscopy the left vocal cord was found immovable in the paramedian position, slightly shortened and excavated. The right vocal cord was freely movable. The voice was slightly hoarse.

Patient no. 38

P. P. Male. Age 54 years.

Cause of vocal cord paresis: Carcinoma of the esophagus.

On April 19, 1954, the patient suddenly became hoarse. On laryngoscopy the right vocal cord was found immovable in the paramedian position.

X-ray examination of the mediastinum showed no abnormality.

X-ray investigation of the esophagus with contrast medium demonstrated a large tumor opposite the bifurcation of the trachea. Esophagoscopy demonstrated a large ulcerated tumor opposite the bifurcation of the trachea. The biopsy diagnosis was carcinoma solidum.

Neurological investigation showed no abnormality.

Wassermann reaction: negative.

Complete blood investigation: no abnormality.

No abnormality on palpation of the neck.

On May 1, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the right vocal and right cricothyroid muscles but not in the left vocal or left cricothyroid muscles. The action potential amplitude in the right vocal muscle was about one third that in the left vocal muscle.

On laryngoscopy the right vocal cord was found immovable in the paramedian position, not shortened or excavated. The left vocal cord was freely movable. The voice was slightly hoarse.

Patient no. 58

C. M. Male. Age 67 years.

Cause of vocal cord paresis: Carcinoma of the lung.

On November 29, 1951, the patient suddenly became hoarse. Laryngoscopy demonstrated the left vocal cord immovable in the paramedian position.

X-ray examination of the lungs and mediastinum demonstrated an infiltration 2 cm in length along the arch of the aorta. Tomography demonstrated the infiltration to lie in the left main bronchus, 3–4 cm inferior to the carina.

Bronchoscopy demonstrated granulation tissue a few cm beyond the bifurcation in the left main bronchus. Biopsy revealed a carcinoma solidum.

Neurological investigation: no abnormality.

Wassermann reaction: negative.

Complete blood investigation: no abnormality.

On August 14, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the left vocal muscle but not in the right vocal or right or left cricothyroid muscles. The action potential amplitude in the left vocal muscle was about half that in the right vocal muscle. The action potential amplitude in the right and left cricothyroid muscles was the same.

On laryngoscopy the left vocal cord was found immovable in the paramedian position, not shortened or excavated. The right vocal cord was freely movable. The voice was somewhat hoarse.

Patient no. 62

W. J. Male. Age 30 years.

Cause of vocal cord paresis: a low trauma to the neck.

On August 12, 1954, the patient received a blunt trauma on the lower half of the neck on the left when he became wedged in a machine. An hematoma developed and his voice became hoarse. On laryngoscopy the left vocal cord was found immovable in the paramedian position.

X-ray examination of the mediastinum showed no abnormality.

Neurological investigation showed no signs of neurological involvement.

Complete blood investigation: no abnormality.

Wassermann reaction: negative.

There was an hematoma the size of the palm of the hand low in the neck on the left side.

On August 26, 1954, electromyography of the laryngeal muscles showed denervation potentials and evidence of loss of motor units in the left vocal muscle but not in the right vocal muscle or right or left cricothyroid muscles. The action potential amplitude in the left vocal muscle was about one third that in the right vocal muscle. The amplitude in the right and left cricothyroid muscle was the same.

On laryngoscopy the left vocal cord was found immovable in the paramedian position, slightly shortened but not certainly excavated. The right vocal cord was freely movable. The voice was weak and hoarse.

Patient no. 24

S. H. Female. Age 57 years.

Cause of vocal cord paresis: unknown.

Since childhood the patient had a slightly hoarse voice. She did not know the cause of this. She remembers particularly nothing about hoarseness developing after a febrile illness, poisoning or exposure to cold. In 1940 laryngoscopy revealed the left vocal cord immovable in the paramedian position.

X-ray examination of the mediastinum: no abnormality.

Neurological examination: no abnormality.

Wassermann reaction: negative.

Complete blood investigation: no abnormality.

No abnormality on palpation of the neck.

On March 6, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the left vocal but not in the right vocal or right or left cricothyroid muscles. The action potential amplitude in the left vocal muscle was about one fifth that in the right vocal muscle. The amplitude in the right and left cricothyroid muscles was the same.

Laryngoscopy demonstrated the left vocal cord immovable in the paramedian position, not shortened or excavated. The right vocal cord was freely movable. The voice was slightly hoarse.

Patient no. 28

G. B. Female. Age 50 years.

Cause of vocal cord paresis: unknown.

On December 23, 1953, the patient became hoarse after an afebrile cold with purulent nasal secretion. Laryngoscopy demonstrated the right vocal cord immovable in the paramedian position.

X-ray examination of the mediastinum and the esophagus showed no abnormality.

Neurological examination: no abnormality.

Wassermann reaction: negative.

Complete blood investigation: no abnormality.

No abnormality on palpation of the neck.

On March 20, 1954, electromyography showed no evidence of loss of motor units in the left cricothyroid, arytenoid and posterior crico-arytenoid muscles.

Laryngoscopy demonstrated the right vocal cord immovable in the paramedian position, slightly shortened but not excavated. The left vocal cord was freely movable. The voice was slightly hoarse.

Patient no. 33

T. G. C. Male. Age 71 years.

Cause of vocal cord paresis: unknown.

Does not remember ever having been hoarse. Is not hoarse now. In 1946 laryngoscopy revealed the right vocal cord immovable in the paramedian position.

No abnormality on palpation of the neck.

The patient refused further investigation.

On April 3, 1954, electromyography of the laryngeal muscles showed no evidence of loss of motor units in the right and left vocal or the right and left cricothyroid muscles. The action potential amplitude in the right and left vocal muscles and in the right and left cricothyroid muscles was the same.

On laryngoscopy the right vocal cord was found immovable in the paramedian position, not shortened or excavated. The left vocal cord was freely movable. The voice was not hoarse.

Patient no. 22

J. L. Female. Age 58 years.

Cause of vocal cord paresis: Tumor of the basal cranium.

In 1947 the patient was operated for a right sided tumor of the jugular globe in the middle ear. There was a recurrence in 1952. Hoarseness since October, 1952. X-ray examination demonstrated that the tumor had invaded the basal cranium and occupied the right jugular foramen. Laryngoscopy revealed the right vocal cord immovable in the intermediate position.

X-ray examination of the mediastinum: no abnormality.

Neurological examination revealed paresis of cranial nerves IX, X, XI and XII on the right side.

Wassermann reaction: negative.

Complete blood investigation: no abnormality.

No abnormality on palpation of the neck.

On February 27, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the right vocal cricothyroid and posterior crico-arytenoid muscles but not in the left vocal and cricothyroid muscles. The action potential amplitude in the right vocal and cricothyroid muscles was about one third that in the left vocal and cricothyroid muscles.

Laryngoscopy demonstrated the right vocal cord immovable in the intermediate position, slightly shortened and excavated. The left vocal cord was freely movable. There was considerably decreased sensibility on the right side of the larynx. The voice was very hoarse.

Patient no. 36

F. D. Male. Age 61 years.

Cause of vocal cord paresis: Metastatic cancer of the basal cranium.

Hoarse since the beginning of March, 1954. In the end of March, 1954, biopsy from the left middle ear demonstrated a plano-cellular carcinoma. Laryngoscopy revealed the left vocal cord immovable in the intermediate position.

X-ray examination of the mediastinum: no abnormality.

Neurological examination demonstrated paresis of the left palatal arch.

Wassermann reaction: negative.

Complete blood examination: no abnormality.

No abnormality on palpation of the neck.

On April 10, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the left vocal and cricothyroid muscles but not in the right vocal and cricothyroid muscles. The action potential amplitude in the left vocal and cricothyroid muscles was about one half that in the right vocal and cricothyroid muscles.

On laryngoscopy the left vocal cord was found immovable in the inter-

mediate position, slightly shortened and excavated. The right vocal cord was normally movable. There was decreased sensibility of the left side of the larynx. The voice was very hoarse.

Patient no. 49

K. J. Male. Age 59 years.

Cause of vocal cord paresis: Cancer metastases to the basal cranium.

Hoarse since April 28, 1954. Laryngoscopy demonstrated the left vocal cord immovable in the intermediate position.

X-ray examination demonstrated a large tumor in the left upper lung field.

Neurological examination suggested increasing strangulation of the left internal carotid artery with cerebral anemia.

Wassermann reaction: negative.

Complete blood investigation: no abnormality.

No certain abnormality on palpation of the neck.

On July 24, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the left cricothyroid and arytenoid muscles but not in the right cricothyroid muscle. The left vocal muscle showed only movement artefact. The action potential amplitude in the left cricothyroid musle was about one third that in the right cricothyroid muscle.

Laryngoscopy revealed the left vocal cord immovable in the intermediate position, slightly shortened and excavated. The right vocal cord was normally movable. There was perhaps slightly diminished sensibility on the left side of the larynx. The voice was very hoarse.

On September 7, 1954, the patient expired. Autopsy demonstrated a large carcinoma in the left upper lung with glandular metastases in the mediastinum, along the internal carotid artery extending up to the basal cranium, as well as multiple metastases in the cerebrum and cerebellum.

Patient no. 60

B. E. A. Male. Age 42 years.

Cause of vocal cord paresis: a high trauma to the neck.

The patient had been hoarse since strangulation in 1950. Laryngoscopy demonstrated the right vocal cord immovable in the intermediate position.

X-ray examination of the mediastinum showed no abnormality.

Neurological examination: no abnormality.

Wassermann reaction: negative.

Complete blood investigation: no abnormality.

No abnormality on palpation of the neck.

On August 21, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the right vocal and cricothyroid muscles but not in the left vocal, cricothyroid and arytenoid muscles. The action potential amplitude in the right vocal and cricothyroid muscles was about one fourth that in the left vocal and cricothyroid muscles.

Laryngoscopy demonstrated the right vocal cord immovable in the intermediate position, excavated but not shortened. The left vocal cord was freely movable. There appeared to be decreased sensibility in the right half of the larynx. The voice was somewhat hoarse.

Patient no. 3

P. M. M. Male. Age 20 years.

Cause of vocal cord paresis: unknown.

In the end of November, 1953, the patient had an afebrile acute tonsillitis lasting several days. He was hoarse thereafter. Laryngoscopy revealed the left vocal cord immovable in the intermediate position.

X-ray examination of the mediastinum: no abnormality.

Neurological examination: no abnormality.

Wassermann reaction: negative.

Complete blood examination: no abnormality.

No abnormality on palpation of the neck.

On December 12, 1953, electromyography of the laryngeal muscles showed loss of motor units in the left vocal cricothyroid muscles but not in the right vocal and cricothyroid muscles. The action potential amplitude in the left vocal and cricothyroid muscles was about one twentieth that in the right vocal and cricothyroid muscles.

On laryngoscopy the left vocal cord was found immovable in the intermediate position, slightly excavated but not shortened. The right vocal cord was normally movable. There was diminished sensibility of the left side of the larynx. The voice was very hoarse.

On February 1, 1954, there were beginning movements of the left vocal cord. On March 14, 1954, the left vocal cord was normally movable.

Patient no. 37

K. N. Female. Age 62 years.

Cause of vocal cord paresis: unknown.

The patient had been hoarse since May 1953. Does not know anything about the etiology. Remembers particularly nothing about the hoarseness having begun after febrile illness, poisoning or exposure to cold. Laryngoscopy revealed the left vocal cord immovable in the intermediate position.

X-ray examination of the mediastinum: no abnormality.

Neurological examination: paresis of the muscles of the tongue on the left side.

Wassermann reaction: negative.

Complete blood examination: no abnormality.

No abnormality on palpation of the neck.

May 1, 1954, electromyography of the laryngeal muscles showed evidence of loss of motor units in the left vocal and cricothyroid muscles but not in the right vocal and cricothyroid muscles. The action potential amplitude in the left vocal and cricothyroid muscles was about one third that in the right vocal and cricothyroid muscles.

On laryngoscopy the left vocal cord was found immovable in the intermediate position, slightly shortened and excavated. The right vocal cord was freely movable. There appeared to be diminished sensibility on the left side of the larynx. The voice was very hoarse.

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